SYPHILITIC DISEASES
OF THE
SPINAL CORD.

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SYPHILITIC DISEASES

OF THE

SPINAL CORD.

BY

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PREFACE.

IN the following pages I have endeavoured to present a more detailed account of spinal syphilis than is generally found in text books or systems of medicine. I have included a number of clinical and pathological observations, which I have made during the last ten years, whilst holding the post of Medical Registrar at the Manchester Royal Infirmary. Some of the cases referred to have been my own patients, but the majority have been in-patients at the Royal Infirmary under the care of the Physicians—Drs. Leech, Dreschfeld, Steell, Harris, Bury, and Wilkinson. To these gentlemen I am indebted for kindly allowing me to record any points of clinical or pathological interest which I may have observed in the cases under their care.

With one exception, the illustrations have all been reproduced from my own drawings. To the kindness of Professor Homén, of Helsingfors, Finland, and Herr W. Braumüller, of Vienna, I am indebted for the illustration on page 25.

I have further to acknowledge my indebtedness to many monographs and articles on Spinal Syphilis, especially to those by Sir William Gowers, Hutchinson, Erb, Oppenheim, Leyden, Goldschieder, Sottas, and others.

The book is devoted to the spinal affections produced by *acquired* syphilis. As the very rare spinal diseases due to hereditary syphilis have not come under my observation, all reference to them has been omitted. The reader will find them described in the writings of G. de la Tourette and others.

In the pathology of spinal syphilis, I have endeavoured to emphasise the importance of vascular changes in many forms of the affection.

The more carefully the morbid anatomy of spinal syphilis is studied, the more clearly do we realise the truth of the views of Dr. Hughling Jackson (which had special reference to the brain, but which apply to all parts of the nervous system), that morbid changes so very frequently do not begin in the nervous elements, but in other structures, such as blood-vessels and connective tissue.

R. T. W.

CONTENTS.

SYPHILITIC DISEASES OF THE SPINAL CORD.

SYPHILIS may produce various clinical forms of spinal disease, and the differential diagnosis from other spinal affections is of much importance. From a pathological point of view, no doubt it might be considered more scientific to describe all the forms together, but from the practical standpoint of clinical diagnosis, it appears more advisable, after a general survey of the etiology, pathology, and diagnosis, to describe the symptoms and morbid anatomy of the various forms separately.

I. ETIOLOGICAL CONSIDERATIONS.

Frequency.—Cases of syphilitic disease of the spinal cord, excluding locomotor ataxia, are somewhat rare. At the Manchester Royal Infirmary, during the ten years I have held the post of Medical Registrar, the following figures will show the frequency of the diseases in relation to the number of cases of nervous disease, &c. :—

No. of medical in-patients in 10 years.	No. of cases of nervous diseases.	No. of cases of locomotor ataxia.	No. of cases of spinal syphilis.
14,575	2,456	118	32

Erb thinks that locomotor ataxia is ten times more common than spinal syphilis.

Boulloche found, in an analysis of 1,085 cases of syphilis of the nervous system, mostly under the care of

Fournier, that there were 77 cases of spinal, and 416 of cerebro-spinal syphilis.

Gerhardt thinks the proportion of cases of spinal to cerebral syphilis to be about 1 : $4\frac{1}{2}$.

There can be no doubt that spinal syphilis is less frequent than cerebral or cerebro-spinal syphilis, and pure spinal cases, without any cerebral symptoms, are somewhat rare. In the cases in which the symptoms have been spinal only, it is possible that a pathological examination would often have revealed slight cerebral changes also. But if we include under the term spinal syphilis, the cases in which there are spinal symptoms only, and also spinal cases in which cerebral symptoms, if present, are slight and of minor importance, then I do not think the disease is so rare as some writers suppose.

Slight or temporary cerebral symptoms are often present in spinal syphilis, such as nocturnal headache, diplopia or strabismus, but this is not always the case. In 11 out of the last 31 cases of spinal syphilis which have come under my observation, there have been cerebral symptoms (usually slight) at some period of the illness.

Sex.—A striking fact, shown by all statistics, is the much greater frequency of the disease in males than females. In the last 31 cases which have come under my observation, there have been 26 males and 5 females. Sottas thinks the proportion of 1 female to 10 males is a fair statement.

It is to be remembered that syphilis is more common amongst males, and this may account in part, but not entirely, for the great difference in the liability of the two sexes to suffer from spinal symptoms.

Age.—Naturally spinal syphilis is most frequent between the 20th and 40th years of age.

In 31 Manchester cases the ages were as follows :—

11	cases	between	20	and	30	years.	
14	,,	,,	30	,,	40	,,	
5	,,	,,	40	,,	50	,,	
1	,,	over	50	years.			
31							

Date of Onset.—The following are the dates of onset in 27 cases in Manchester :—

Date after Infection.	*Cases.*
Under 12 months.....	2
About 1 year.....	1
1 to 2 years	3
About 2 years	2
2 to 3 years	1
About 3 years	2
,, 4 ,, ...	4
,, 5 ,, ...	2
,, 6 ,, ...	1
,, 7 ,, ...	1
,, 8 ,, ...	1
,, 9 ,, ...	1
,, 13 ,, ...	1
,, 18 ,, ...	1
,, 19 ,, ...	2
,, 25 ,, ...	1
,, 27 ,, ...	1
	27

These figures show that the disease may occur at almost any date after infection, but more than half (17 out of 27) occurred during the first five years.

In 71 cases Boulloche found that 62 per cent. occurred during the first four years.

According to Oppenheim most cases occur within the first six years.

Spinal syphilis may develop within the first twelve months of infection (as shown by the table just given). I have observed it in one case as early as seven months after primary syphilis; in another of the cases tabulated (page 3), it occurred within the first twelve months; and a number of similar cases are on record. Goldflam gives the following dates of onset after the primary infection in eighteen cases:—In the first six months, four cases; in the second six months, two cases; after one year, three cases; in the course of the second year, four cases; more than two years after infection, five cases.

On the other hand, some cases occur long after infection (ten or fifteen years). In both the early and the late cases, often the clinical history and pathological examination leave no doubt as to the syphilitic nature of the spinal changes.

Relation to nature and treatment of early syphilis.—In the cases which I have observed, more frequently the early symptoms of syphilis have been very slight; but in some they have been of moderate or great severity. As regards the nature of the early antisyphilitic treatment, it is difficult to draw conclusions in all cases, but certainly in the majority it has only been continued a very short time. In a few of the cases the patient had received antisyphilitic treatment for a long period after the infection, though of course, no information could be obtained as to the actual doses of the drugs.

Many writers have drawn attention to the slight nature of the early syphilitic symptoms, and to the absence of thorough antisyphilitic treatment at an early stage, when the patient has suffered from spinal syphilis at a later date. One may conclude that the slight nature of the early

syphilitic symptoms does not exclude the possibility of spinal syphilis later, but it is difficult to prove that in such cases spinal disease is more likely to follow.

Predisposing Causes.—Occasionally spinal syphilis has followed injury to the back, or exposure to cold, but certainly this history has been rare in the cases which have come under my observation, and it is very doubtful what importance ought to be attached to such antecedents.

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(3.) Goldflam.—*Ueber Ruckenmark Syphilis, Wiener Klinik*, 1893.

II. GENERAL CONSIDERATIONS RESPECTING THE PATHOLOGICAL ANATOMY AND CLINICAL FORMS OF SPINAL SYPHILIS.

Syphilis may produce various pathological spinal changes, of which the following are the most important:—(*a*) Diseases of the blood vessels—endarteritis and periarteritis, endo-phlebitis and peri-phlebitis: (*b*) Partial or complete obstruction of the blood vessels by thrombosis or thickening of the vessel wall, and changes resulting therefrom, *e.g.*—softening, degeneration, hæmorrhages. (*c*) Inflammation of the meninges or of the cord. (*d*) Gummatous infiltration of the cord or membranes. (*e*) True circumscribed gummata of the cord or membranes. (*f*) Sclerosis secondary to destruction of nerve elements produced by the processes previously mentioned. (*g*) A chronic post-syphilitic degeneration—locomotor ataxia.

The pathological changes which indicate syphilis most clearly are, gummata, gummatous nodules in the walls of the vessels, inflammation of the meninges followed by caseous and fibroid changes. The arterial changes, endarteritis and periarteritis are not quite conclusive evidence of syphilis, since they have been found in non-specific cases; but as Ziegler points out in the latter, there is not such a marked tendency to cell proliferation in the arterial coats, especially in the adventitia, as in true syphilitic disease.

It is to be hoped, that in the future bacteriological research will lead to the discovery of a characteristic micro-organism in syphilitic lesions.

In those cases of spinal syphilis in which gummata, gummatous arteritis, gummatous meningitis, or infiltration of the cord are not present, it is sometimes difficult, simply from the microscopical examination of the cord, to be certain of the syphilitic origin of changes of the nature of inflammation or softening. Thickening of the vessel walls and proliferation of the endothelium of the intima, as just stated, may be produced by other cause besides syphilis. Still syphilis is a very common cause of endarteritis. If the myelitic or degenerative changes should be associated with marked endarteritis and thickening of the vessel walls, and if there should be a history of syphilis, but no evidence of any of the other common causes of the vascular changes mentioned, then there is, of course, the strongest probability that the spinal affection is specific.

In a few cases of spinal lesions occurring in individuals who have suffered previously from syphilis, the pathological changes have been almost identical with those found in non-syphilitic myelitis, and the endarteritis and marked thickening of the arteries have been absent. Whether such myelitic changes are always due to syphilis is questionable. It is quite possible that some of the cases have been simply instances of non-specific myelitis, occurring in individuals who happen to have previously suffered from syphilis. In a large proportion of cases of spinal syphilis, both acute and chronic, arterial changes are very prominent, and it appears extremely probable that they are the cause of the changes in the nerve elements.

Naturally the clinical features of spinal syphilis will vary according to the nature, extent, and localisation of the pathological changes, and the following table shows

the chief clinical varieties, with their associated pathological lesions :—

FORMS OF SPINAL SYPHILIS.

Clinical Forms :—	*Associated Pathological Conditions* :—
1. Syphilitic disease of the vertebræ.	Syphilitic caries, necrosis, gummata, exostoses, periostitis and osteitis of vertebræ.
2. Chronic syphilitic meningitis.	Chronic pachymeningitis, chronic leptomeningitis, or both combined,
3. Meningo-myelitis.	Leptomeningitis with invasion of the cord ; the proportion of the meningitic and myelitic changes varying in different cases. Often small gummatous infiltrations of the meninges and of the cord. Vascular changes often well marked.
4. Paraplegia of acute onset (so-called acute syphilitic myelitis.)	(*a*) Softening due to vascular disease ; thickening of vascular walls and narrowing or obliteration of lumen. (*b*) Vascular disease with thrombosis and secondary hæmorrhage or softening. (*c*) Myelitis without thickening of vessel walls or thrombosis. Changes resemble non-specific myelitis.

5. Chronic syphilitic spinal paralysis (Erb's spinal syphilitic paralysis).	Scattered syphilitic changes chiefly in the white matter and periphery of the cord, associated with vascular disease and slight meningitis.
6. Meningeal and intramedullary spinal tumour.	Gummata of meninges, or of the cord.
7. Anomalous Forms :	
a. Brown-Séquard's paralysis (hemiparaplegia).	Unilateral lesion of the cord : Gummatous meningomyelitis.
b. Triplegia (both legs and one arm paralysed—hemiplegia and paraplegia).	Combined unilateral cerebral lesion, with bilateral spinal lesion.
c. Cases simulating disseminated sclerosis.	Multiple syphilitic lesions.
d. Cases simulating primary lateral sclerosis.	Sclerosis of crossed pyramidal tracts of the spinal cord with slight changes elsewhere.
e. Cases simulating anterior poliomyelitis.	(?) Probably lesion of, or in distribution of, central arteries of spinal cord—anterior median arteries.
f. Cases simulating amyotrophic lateral sclerosis.	(?) Meningo-myelitis in lower cervical region.
g. Case simulating pseudo-hypertrophic paralysis and idiopathic muscular atrophy, in the gait and manner of rising into the erect posture.	(?) Meningitis at the lower part of the cord.

h. Cases simulating syringomyelia as regards sensory symptoms.	Meningo-myelitis.
i. Cases presenting symptoms of meningo-myelitis during life.	Pathologically simulating syringo-myelia: meningo-myelitis with cavities in the grey matter of the cord.
j. Cases simulating locomotor ataxia (syphilitic pseudo-tabes).	Meningo-myelitis invading the posterior columns: gummatous infiltration of the posterior columns.
8. Post syphilitic degeneration—locomotor ataxia.	Sclerosis in the posterior columns. Changes in posterior nerve roots, &c.

Form of Spinal Syphilis in 32 Cases.

In the last 32 cases of spinal syphilis, which have come under my observation in Manchester, the forms of the disease were as follows:—

	CASES.
Syphilitic disease of the vertebræ	0
Chronic syphilitic meningitis	3
Meningo-myelitis	16
Acute paraplegia—"acute myelitis"	6
Chronic syphilitic spinal paralysis (Erb's)	4
Tumour (gumma) of the cord	1
Triplegia	1
Pseudo-tabes	1
	32

III. GENERAL REMARKS ON THE DIAGNOSIS OF SPINAL SYPHILIS.

The diagnosis of spinal syphilis is of great importance, especially at the early stage, when by vigorous treatment we may hope to prevent further destruction to nerve structures.

Not infrequently in spinal syphilis, well marked symptoms are present for a long period before the cause of the disease is recognised. Thus valuable time is lost before anti-syphilitic treatment is commenced.

In spinal disease of obscure origin, it is far better, if there should be the slightest possibility of previous syphilis, to give the patient the benefit of the doubt, in the pathological sense, and to employ anti-syphilitic treatment at once.

Before describing the various forms of spinal syphilis, it may be of service to consider, in a general manner, the points in the clinical history and symptomatology on which the diagnosis of the syphilitic nature of spinal diseases may be based. Spinal syphilis may simulate various forms of non-syphilitic diseases of the spinal cord, but the differential diagnosis of each special variety will be afterwards considered separately.

The following are the most important points in favour of the syphilitic nature of a spinal disease :—

(1.) *History of previous Syphilitic Infection.*—The history of a hard chancre followed by secondary symptoms is very important. As already stated, the primary and secondary symptoms may have been very slight, and

sometimes they have been entirely forgotten. Sometimes no history can be obtained, and yet the clinical course, or the autopsy, shows that the case is undoubtedly syphilitic. In two cases of spinal syphilis which have come under my observation, the patients have denied syphilis, and yet on inspecting the soft palate, I have found a syphilitic perforation, and the other symptoms and clinical course have left no doubt as to the nature of the spinal affection. If a syphilitic history cannot be obtained, we are not justified in concluding that the case is not specific. As Sir Wm. Gowers remarks, "we cannot absolutely exclude syphilis unless we can exclude the possibility of infection." On the other hand, it does not follow that a spinal disease is syphilitic because the patient happens to have suffered previously from syphilis. The frequency of syphilis amongst persons not suffering from spinal disease is to be taken into account, and this varies no doubt in different localities. From a consideration of statistics published, Gowers thinks 13 per cent. of the adult male population in London have suffered from syphilis, and 8 per cent. in Leeds. These figures have to be taken into account when considering the frequency of a syphilitic history in any class of spinal affections.

(2.) *Signs of present or previous Syphilitic Disease in various parts of the body.*—Active syphilitic processes may be present in other parts of the system at the onset of the spinal symptoms. Thus, for example, in 3 of the 32 cases mentioned on page 11, there was a slight rash of copper-coloured patches on the skin, at the time that the patient first came under treatment for the spinal symptoms. In two other cases (mentioned already,) there was ulceration and perforation of the soft palate.

Evidence of previous syphilis is very important when the history of venereal disease is denied, or when the

patient is indefinite on this point. It is not possible to mention all of the well-known indications of previous syphilis, but the following, amongst others, are of great importance:—Cicatrix on the penis; cicatrices of old ulcers of circular shape on the legs or face; cicatrices on the throat or soft palate, or old perforation of the latter; periosteal nodes. The eye often presents valuable indications; for example, irregularities of the pupil, adhesion of the iris to the lens capsule and other evidences of old iritis, immobility of the pupil, choroiditis, choroido-retinitis, syphilitic retinitis, fine opacities of the vitreous.

(3.) *The presence of Cerebral Symptoms.*—In many cases of spinal syphilis, no cerebral symptoms are present. In others, however, though the symptoms are chiefly spinal, there are indications of implication of the brain also. Nocturnal syphilitic headache is a common cerebral symptom. Other symptoms are diplopia, paresis or paralysis of one or more ocular muscles, immobility of the pupils, facial paralysis, hemianopsia, trigeminal neuralgia and anæsthesia, neuro-paralytic ophthalmia. More severe symptoms are headache, with vomiting and optic neuritis. Hemiplegia may develop, and if paraplegia was already present, then three limbs will be paralysed—a condition known as triplegia. Epileptic attacks, unilateral or general, may also occur. The symptoms mentioned are, of course, due to associated syphilitic disease of the brain (meningitis, gummata, thrombosis, &c.)

As regards the actual *spinal* symptoms, there are none which are absolutely diagnostic, but the following points in the symptomatology are in favour of the syphilitic nature.

(4.) *The relatively slight intensity of the cord disease as compared with the extensive area involved,* (Sachs.)—The

irregular extension and incomplete nature of the spinal lesions are very suggestive of syphilis.

(5.) Often there is present at some period of the illness, *Brown-Séquard's group of symptoms* of unilateral lesion of the cord (paralysis of one leg, and anæsthesia of the other). Usually it is an incomplete form however, one leg being simply more paralysed than the other, and the sensation being most affected in the leg which is least paralysed. As a rule, the difference rapidly passes away, or greatly diminishes, and soon both legs are almost equally affected.

(6.) Another point to which importance has been attached, is the peculiar *fluctuation of the symptoms.* Remission and relapses occur, and there are variations and oscillations in the intensity of the individual symptoms. The paralysis and anæsthesia advance in outbursts or stages, and there are often spontaneous remissions.

Oppenheim attaches importance to the variations in the knee jerks which are observed in certain cases. At one time the knee jerk may be present, a little later absent, and then present again. I have observed these variations in the knee jerk, but not very often. The return of the pupillary reflex after it has been once lost is also of some importance.

(7.) In some cases, but not in all, the symptoms indicate a *multiplicity of lesions.*

(8.) Symptoms of meningitis and of irritation of spinal nerve roots are in favour of syphilis, such as stiffness of the back, pain in the back, radiating pains and hyperæsthesia in the limbs, girdle sensations.

Much importance has been attached by French authors to pain in the back which is worse at night. This symptom I have observed in a number of cases.

(9.) Gowers states that "as a general rule, if we find certain structures of common function picked out for isolated impairment from among others of different function, we may be sure that we have not to deal with a true specific process. It may be a post-syphilitic degeneration, but it is not a specific lesion."

(10.) *Improvement under anti-syphilitic treatment* is of of some value, but, of course, is not conclusive; since in some cases, undoubtedly due to syphilis, anti-syphilitic treatment has no effect; whilst on the other hand, cases which are not syphilitic may recover under this treatment. Gowers very clearly points out the caution which ought to be observed in drawing a conclusion from the effects of treatment in all cases of syphilis of the nervous system:—

"(*a*). The effect of the drug must be decided and unequivocal.

(*b*). Its influence must not be obscured by any other change in the measures employed that might cause the improvement observed.

(*c*). The lesion must not be one of which the symptoms tend to subside spontaneously.

(*d*). The lesion must not be one that can be influenced by the drug when it is not due to syphilis.

(*e*). Before a negative result can be admitted as evidence that the lesion is not due to syphilis, we must be satisfied that the damage produced is not so great that its manifestations *cannot* speedily subside."

"The method is applicable chiefly to chronic lesions in which the influence of treatment can be clear and decided. Acute lesions, if they are slight, have too great a tendency to subside, if they are severe, have effects of prolonged duration."

References.

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(3.) Oppenheim.—*Lehrbuch der Nervenkrankheiten*. Berlin, 1898.

(4.) Sachs.—*Brain*, 1893, p. 414.

IV. SYPHILITIC DISEASE OF THE VERTEBRÆ.

Syphilis frequently causes diseases of the bones of the limbs, of the skull, of the clavicle, &c., but it is remarkable that it comparatively rarely gives rise to any affection of the vertebræ. A few cases of syphilitic disease of the vertebræ are on record, however, in medical literature. In some of these cases there were no signs characteristic of syphilitic disease, and the diagnosis was based (1) on evidences of disease of the vertebræ occurring in patients who had had syphilis, (2) on the improvement under antisyphilitic treatment. It is possible that some of them have been cases of ordinary tubercular caries in persons having a syphilitic history. In other cases, however, there have been indications of the syphilitic nature of the vertebral disease, or an autopsy has shewn the diagnosis of syphilitic disease to be correct.

The vetebral disease may be the result of the extension of syphilitic disease of the bones of the skull down to the spinal column; or it may be the result of syphilitic ulceration of the throat extending deeply and involving the cervical vertebræ; or the syphilic bone disease may originate primarily in the vertebræ, and sometimes it appears to be excited by injury.

Pathologically, the following forms of specific bone disease have been met with in the vertebræ:—

(1) Syphilitic periostitis; (2) gummata in the bone or on the surface thereof; gummatous osteitis; (3) syphilitic caries; (4) syphilitic necrosis: (4) syphilitic exostoses on the bodies of the vertebræ and their processes.

Symptomatology.—As a result of the exostosis, caries, gummata or other form of bone disease, the nerve roots may be compressed; or pachymeningitis or compression myelitis may follow. But disease of the spinal cord or membranes, owing to syphilic bone disease, is very rare.

Oppenheim mentions a case in which large exostoses of irregular form developed in the upper cervical vertebræ and could be felt in the neck. Paralysis and sensory disturbances developed in all four extremities, along with signs of a cervical myelitis. There was a definite syphilitic history and an energetic long mercurial treatment was followed by recovery.

Oppenheim mentions another case in which a round exostosis, the size of an apple, situated about the tenth or eleventh dorsal vertebra, caused marked signs of compression of the cord. In this case also mercurial treatment was followed by recovery.

Gummatous tumours may also develop in the bone or periosteum and cause pressure symptoms.

A syphilitic ulcer of the throat has been known to extend deep, and involve and destroy a cervical vertebra. Thus Autenreith (quoted by Leyden and Goldscheider) reports the case of a man aged 20, who suffered from syphilitic ulceration of the fauces. This invaded the vertebræ and exposed the spinal dura-mater, so that it could be seen through the mouth. After death the anterior arch of the atlas was found destroyed, and the anterior surface of the odontoid process was affected.

Syphilis may produce softening and destruction of the vertebræ—a caries resembling that produced by tuberculosis; and this may he followed by deformity and curvature of the spinal column just as in tubercular caries. Even when the vertebral disease is considerable, the spinal cord may remain unaffected. But in other cases

symptoms of compression of the nerve roots of the spinal cord develop, as in compression from tubercular caries. The root symptoms are sometimes very marked. When a "compression myelitis" is produced the symptoms vary according to the position of the diseased vertebræ. When the cervical vertebræ are affected there is paralysis of all four limbs; when the dorsal or dorso-lumbar region of the vertebral column is diseased, the legs are paralyzed. Bladder and rectal symptoms and bedsores often develop, and there may be sensory symptoms also. The paralysed legs are generally spastic, but flaccid if the lumbar enlargement is compressed.

In many cases, the syphlitic vertebral disease gives rise to pain and difficulty of movement only. There is pain in the region of the disease; the affected vertebræ are tender on percussion, and pains radiate along the nerve trunks coming from the diseased parts. Movements at the affected part of the spinal column are painful and difficult.

Gowers mentions deep-seated thickening of the tissues about the cervical vertebræ, apparently due to syphlitic cellulitis. The swelling may be situated on each side and behind the upper cervical spine or deep in the posterior triangle of the neck. "It may damage the nerves before they enter the brachial plexus, causing a definite palsy, as in one case of the lower arm muscles. Movements of the neck may be interfered with and irritation of the nerves may cause neuralgia-like pain, generally felt down the arm, and often very severe." "The spinal cord does not usually suffer." Gowers states that the symptoms pass away under antisyphilitic treatment. He adds that in one case iodide had no influence, but mercury quickly cured.

The diagnosis of syphilitic disease of the vertebræ is based on the following consideration :

(1) The evidence of disease of the vertebræ, which may or may not be associated with signs of compression of nerve roots or of the spinal cord.

(2) The absence of indications of tubercular caries, of carcinoma, or of other spinal tumour, or of symptoms of tuberculosis or tumour growths in any other part of the body.

(3) A history of previous syphilitic infection and the presence of indications of syphilitic disease (recent or old) in other parts of the body, such as syphilitic ulcers of the skin, plantar psoriasis, nodes on the tibia, syphilitic disease of other bones, &c., &c.

(4) The good effect of anti-syphilitic treatment.

Care must be taken to exclude tubercular caries or malignant disease before giving a diagnosis of syphilitic disease.

In spinal tubercular caries there may be evidence of tubercular disease of lungs, or of other parts of the body ; also there may be an irregular temperature, wasting, and other symptoms of tuberculosis. In cases of malignant disease of the vertebræ, the course of the illness is more rapid ; there is general emaciation ; tumours may be found in other parts of the body, and as a rule the pain is more severe.

Jasinski believes that primary tubercular caries of the spinal vertebræ occurs only in children and young persons, that in adults tuberculosis of the vertebræ is only secondary, and that in such cases tubercular disease of the lungs, larynx, or other parts, is then easily detected. In an *adult* presenting signs of caries of the spine, with deformity, if no evidence of tuberculosis of the internal

organs can be detected, Jasinski thinks the diagnosis rests between new growth and syphilitic disease.

As regards the prognosis, probably many of the slighter forms of syphilitic affection of the vertebræ recover. In 8 cases of syphilitic caries, Jasinski reports that 5 completely recovered, 2 improved markedly, and in one the result was not known.

When a diagnosis of syphilitic disease is very probable, anti-syphilitic treatment should be prolonged, but if the diagnosis be doubtful, it must be employed cautiously, as this treatment might be very injurious, if the case should be one of tubercular caries.

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(7.) Leyden.—*Berliner klin. Woch.*, 1889, No. 21.

(8.) Oppenheim.—*Lehrbuch der Nervenkrankheiten Zweite Auflage*, Berlin, 1898, p. 227.

(9.) Rumpf.—*Die syphilitischen Erkrankungen des Nervensystems*, Wiesbaden, 1887, p. 312.

(10.) Sottas.—*Contribution a l'étude anatomique et clinique des paralysies spinales syphilitiques*, Paris, 1894, p. 21.

In the paper by Jasinski, and in the works of Rumpf and Sottas, abstracts are given of the cases hitherto recorded.

V. MENINGITIS.

Pachymeningitis and Leptomeningitis.—Chronic inflammation of the meninges is a common form of spinal syphilis. Often spinal syphilitic changes first commence in the meninges, and this is probably owing to their great vascularity. Usually meningitis is associated with changes in the spinal cord, and the meningeal and spinal affections vary considerably in extent and relative proportion. Both may be well marked; or the meningeal changes may be extensive and the cord changes slight; or the cord changes extensive and the meningeal changes very slight. The important group of cases of combined affection of the meninges and cord will be considered later in the section devoted to meningo-myelitis. But there is a small group of cases in which syphilis produces meningitis without any implication of the cord; and there are other cases, more numerous, in which the pathological changes in the meninges are so marked, and those in the cord so slight, that they may be classed as meningitis for all practical purposes.

In syphilitic meningitis often all three membranes—pia-mater, arachnoid and dura-mater—are affected. The changes may be well marked in all three membranes and have then probably commenced about the same time in all. In some cases the changes appear to indicate that the arachnoid and pia-mater have been affected first, and the dura-mater secondarily. But there are other cases in which the dura-mater is chiefly affected and greatly thickened, and to these the term pachymeningitis is applied. In such cases the inflammatory changes have usually commenced on the inner side of the dura-mater

(pachymeningitis interna). But occasionally the inflammatory changes commence on the outer side of the dura-mater (pachymeningitis externa), and in these cases the meningeal changes are secondary to syphilitic disease of the vertebræ.

There are also cases in which the inflammatory changes affect the arachnoid and pia-mater whilst the dura-mater is not implicated.

Often the spinal meningeal changes are limited to, or at least much more pronounced in, one region of the cord. It is rare for the meningitis to affect the whole length of the cord, or if this is the case, the changes are usually much more intense at one region. The meningitis is frequently limited to, or much more intense in, the cervical region, and in many of these cases the spinal affection appears to be a downward extension from a chronic cerebral meningitis. But this is certainly not always the case, and often meningitis is chiefly in the lumbar and the scaral regions of the cord or about the cauda equina. Occasionally there is well-marked meningitis from one end of the cord to the other.

At a late stage of syphilitic meningitis, frequently dura-mater, arachnoid and pia-mater are all united together into one fibrous cicatricial sheath closely surrounding the spinal cord. (See fig. 1).

The arachnoid and pia-mater are very vascular; they form, in fact, a vascular net-work around the cord. In spinal syphilis the vessels are liable to suffer at an early stage, and hence changes frequently commence in the vascular meninges. In cases in which the meninges present signs of early or slight inflammation, the vessels usually show well-marked changes in either the external or internal coats (periarteritis or endarteritis) or both.

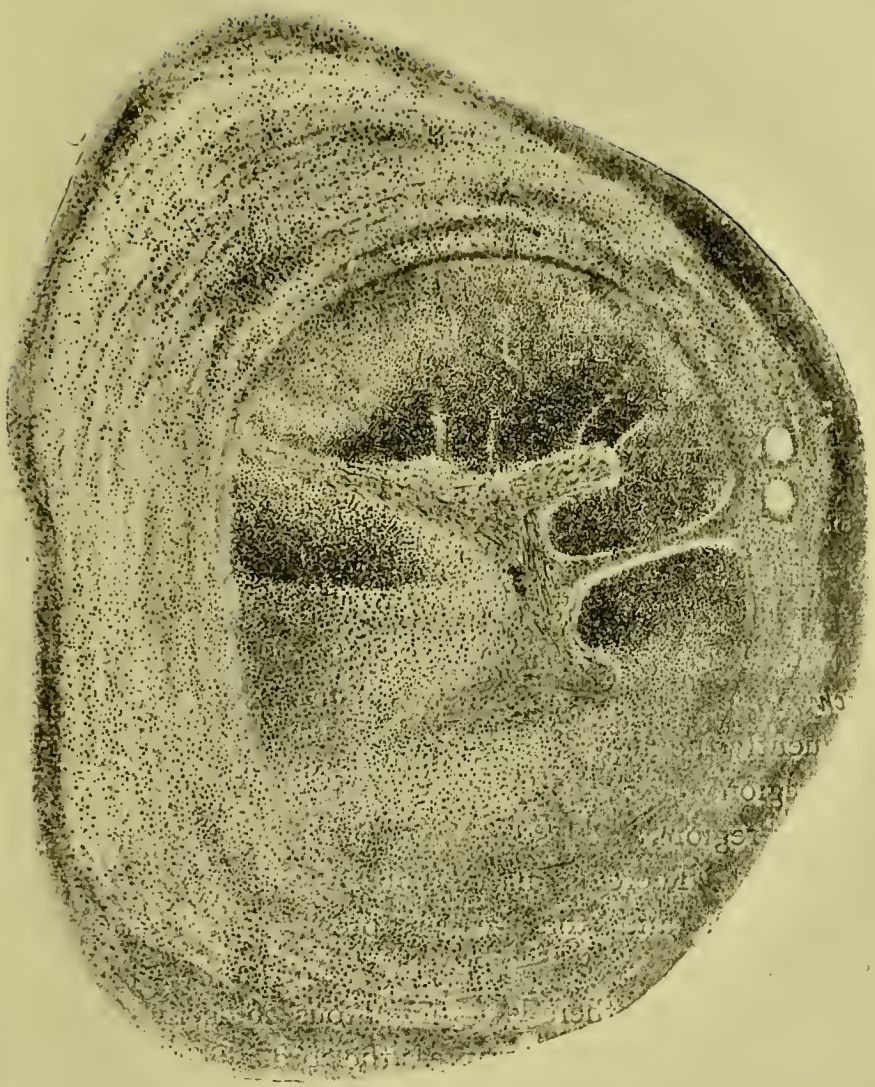

Fig. 1.

Syphilitic Meningitis, dorsal region. Pachymeningitis and leptomeningitis All membranes united to form a fibrous sheath around the cord. (After *Homén. Archiv. f. Dermat. und Syph. Wien.* Bd. xlvi., 1898.)

The early signs of meningitis consist of round-celled infiltration just around the vessels, and where the meningitis is slight the membranes may appear almost normal a short distance from a large blood vessel. Where the meningitis is well-marked, however, there is diffuse cell-

infiltration in the membranes, but the collection of round cells is still most abundant around the larger blood vessels. The meningitis is often more marked posteriorly. At an advanced stage the membranes may present irregular thickenings at various parts, due to fibroid and caseous changes.

In the local form of syphilitic meningitis there is irregular thickening of the pia-mater and arachnoid, and the extensive production of new tissue in the affected region often resembles a diffuse growth as much as a chronic inflammation. "The new tissue has a peculiar gluey aspect, and presents a tendency to caseous degeneration and fibroid change, so that scattered or coalescing cheesy spots are ultimately met with in tracts of mixed fibrous and gelatinous aspect."—(Gowers).

When the meningitis takes the form of *hypertrophic pachymeningitis*, as already mentioned, the cervical region is frequently the seat of the disease; often it is localised to this region, but not always; it may occur in the dorsal or lumbar regions; and sometimes the pachy-meningitis is extensive. There is marked fibrous thickening of the dura-mater, and the vessels present endarteritis or periarteritis.

In other cases there is a gummatous thickening of the dura-mater which may compress the cord. The arachnoid is often involved to a less degree, and the spinal cord may show evidences of incomplete myelitis, softening or gummatous infiltration. The inflammatory changes in the arachnoid and pia-mater may affect all parts of the cord, whilst the pachymeningitis may be localised to one region —cervical, dorsal or lumbar.

Symptomatology—It is important to recognise syphilitic meningitis at an early stage, because the chief blood vessels of the cord run in the meninges, and hence the

cord is very liable to be implicated secondarily if the disease should not be arrested by treatment. Of course, syphilitic disease may commence in the cord itself, and the meningeal affection may be slight and secondary; but in many cases the reverse condition is met with, and the meningeal changes play an important part in the production of the cord lesion. Hence, in practice it is important to remember that spinal meningitis is often syphilitic.

The most prominent symptom of syphilitic spinal meningitis is pain in the back. This is best marked at the region chiefly effected. The pain is often worse at night, like syphilitic headache, and this point has been especially emphasised by French writers. In the cases which have come under my own observation it has often had this character, but in other cases it has been worse during the day. The pain is increased by movement; it often shoots along nerve roots and gives rise to a girdle sensation or to radiating pains in the limbs. There is often rigidity of the spinal column, localised or general, and also tenderness of the spinous processes.

The implication of sensory nerve roots gives rise to pain, and hyperæsthesia in their distribution, and finally impairment of sensation. Implication of the motor roots may produce stiffness of the spinal column, and finally wasting of the groups of muscles supplied by the nerves affected. There are not, however, the paroxysms of muscular spasm which occur so often in acute non-specific meningitis.

When the cervical meninges are affected, there may be unilateral dilatation or contraction of the pupil.

A point of great importance in syphilitic meningitis, is the fact that the temperature is either not raised or only raised very slightly (to 100°F).

The onset of syphilitic meningitis may be sudden or very gradual, and the course is usually chronic. Very frequently the cord becomes implicated, and the affection is then one of meningo-myelitis, but in a few cases the cord is spared, or only affected at a very late period.

When the pathological condition is one of hypertrophic cervical pachymeningitis, there is severe pain in the neck, radiating into the arms. This is followed by hyperæsthesia, spasm of muscles, weakness, diminished sensation, and atrophy of muscles in the arms, especially of the small muscle of the hand when the lower cervical region is affected. In this form the symptoms correspond to those of hypertrophic cervical pachymeningitis as described by Charcot and Joffory. Spastic paresis of the legs may develop; the bladder and rectum may become paralysed; and sensation in the legs and trunk may become impaired.

When syphilitic spinal meningitis is associated with meningitis at the base of the brain, the spinal accessory nerve may be implicated, and paralysis of the sterno-mastoid, of the upper part of the trapezius, and of one vocal cord may be produced; also the hypoglossal nerve may be implicated on one side, and hemiatrophy of the tongue may result.

When the membranes of the lower part of the cord are involved, the patient suffers from pain in the lower part of the back, in the gluteal region, and down the back of the thighs. Occasionally I have noticed paresis of the gluteal muscles which extend at the hips; the patient has had difficulty in rising from the sitting into the erect posture, and the gait has resembled that of pseudo-hypertrophic paralysis (see two cases recorded at the end of this section).

Diagnosis.—The symptoms of syphilitic meningitis

agree in many points with those of the non-syphilitic variety. The history of previous syphilitic infection, or signs thereof, and the absence of fever are important diagnostic indications.

In the differential diagnosis from tubercular meningitis, in favour of the latter, would be the presence of tuberculosis of the lungs or other parts ; but signs of these affections may be absent, especially in children. A family history of tuberculosis, a cachetic or wasted appearance, persistent diarrhœa, tubercles in the choroid on ophthalmoscopic examination would, of course, be in favour of tubercular meningitis. In syphilitic meningitis the temperature is not raised. Lumbar puncture may be of service : the presence of tubercle bacilli in the fluid withdrawn, would be conclusive of tubercular disease.

Syphilitic meningitis has also to be diagnosed from commencing tubercular caries of the spine. In the latter affection deformity (the presence of one or more prominent spinous processes) is of the greatest importance ; but this sign is not always present at an early stage. Tenderness on percussion localised to one or two vertebral spines is in favour of caries ; whilst in syphilitic meningitis, tenderness is more extensive. Also in tubercular caries of the vertebræ, pain is often produced in the back by pressure on the head or on the shoulders.

In acute "idiopathic" meningitis there is elevation of temperature, whilst this sign is absent in syphilitic meningitis ; also the general condition is usually good in the latter affection, whilst it is markedly affected in the former.

In a few cases only, as already mentioned, do the changes remain limited to the membranes. When the affection is diagnosed early, good results may be obtained by anti-syphilitic treatment, but often these cases are not

recognised until the changes have extended beyond the meninges.

The following are notes of three cases of specific meningitis :—

Meningitis. (Lower part of cord). Loss of power in the extensors of the hip, affection of gait and difficulty in rising into erect posture, as in pseudo-hypertrophic paralysis.

Joseph R., aged 33. I first saw the patient in May, 1898, in consultation with Dr. Greenhalgh of Bury. He then complained of pain in the back, and difficulty in walking.

Two years previously he had suffered from pain in the back, chiefly in the lumbar region. This had improved, but some months later, early in 1897, he was again troubled with the pain in the back, and also with pain down the back of the left thigh. After a temporary improvement, he began to suffer from pain at the back of the right thigh in September, 1897. He had also difficulty in walking, and in rising from the sitting to the erect posture.

In November. 1897, he suffered from pain down the *front* of the right thigh, and pain down the *back* of both thighs. In March, 1898, he experienced very great difficulty in rising from the sitting to the erect posture (for six months previously, the pain had been very severe). There had been no bladder symptoms and no girdle sensation.

There was a history of syphilis many years previously. (? about the age of 20), for which he had been treated medically for two years. No history of injury.

On examination, May 26th, 1898, the patient complained of pain at the lower part of the back, in the lower lumbar and sacral regions. The pain passed down the back of the thighs along the course of the sciatic nerves, but there was also much pain down the front of the thighs. There was tenderness on percussion over the sacrum, but no special tenderness on pressure over the course of the sciatic nerves. When the patient stood with his feet together, and the knees extended, and stooped forward, there was pain along the course of the sciatic nerves. Also when the patient was in bed, and the legs were raised, the knees being kept straight, there was pain along the course of each sciatic. There was no disease of the hip joints.

The gait was peculiar, it resembled that of pseudo-hypertrophic paralysis. The body waddled from side to side, and there was lordosis. The patient had great difficulty in getting from the recumbent into the erect posture. He could only perform this movement slowly, and by supporting the weight of his trunk with the hands, on the knees or thighs (just as occurs in pseudo-hypertrophic paralysis). Also he was unable to rise from the sitting into

the erect posture without assistance. The flexors of the hip, the flexors and extensors of the knee and of the ankle acted powerfully on each side. The difficulty in walking and in getting into the erect posture was apparently due to weakness of the gluteal muscles, which extend at the hip joint. There was no anæsthesia in the gluteal region or on the legs; but on the outer side of each leg below the knee, sensation was not quite so sharp as on the inner side of the leg. Knee jerks present, R normal, L very feeble; no ankleclonus; plantar, cremasteric, abdominal and epigastric reflexes present. Pupils reacted to light and accommodation. Ophthalmoscopic examination, fundi normal.

The bladder and rectum, the arms and cranial nerves were not affected. Heart, lungs, and other organs normal.

Under potassium iodide and mercurial inunctions, improvement occurred; the pain diminished, and the walking became less difficult.

Five months later the knee jerks were absent. Flexion of the left thigh (at the hip) was feeble. He was still unable to rise from the sitting into the erect posture without assistance; but when once in the erect posture, he could walk quite well. No ataxia.

A month later the right knee jerk was present, the left very feeble. He had still the same difficulty in rising from the sitting to the erect posture. The pain had occurred from time to time in the legs, but had gradually become less frequent. The temperature was always normal.

The following are notes of a case in which the symptoms were probably due to *meningitis in the upper cervical region.*

E. W., aged 28, consulted me on account of pain in the neck 3½ years after syphilitic infection. (Previously there had been occasional shooting pains in the course of distribution of left ulnar nerve and in the right leg just by the outer malleolus for several weeks). The pain in the neck increased, and was most severe at the upper part, in the region of the first two cervical spines. It was worse at night and radiated up to the occiput. It was of a dull aching character, but there were frequent attacks of sharp shooting pain. There was also tenderness over the upper cervical spines, and over the occipital region. The pain continued about 16 or 17 days and then gradually subsided. A few copper-coloured spots from ¼ to ½ an inch in diameter were present on the arms, and thorax.

Potassium iodide in 10gr. doses caused much gastric disturbance, but the following prescription did not give rise to any discomfort:—

R. Potassii iodidi	℥ii
Spiritûs ammoniæ aromatici	℥iss
Syrupi aurantii	℥vi
Aquam ad	℥vi

Sig. Two *tea*spoonfuls every two hours. A little milk to be taken after each dose of medicine.

The following are the notes of a case in which the symptoms were probably due to *meningitis* at the lower part of the cord.

S. M., aged 36, was admitted as an in-patient at the Manchester Royal Infirmary, under the care of Dr. T. Harris.

He had suffered from syphilis whilst a soldier in India 14 years previously, and had received no internal treatment.

Two years before admission he had begun to suffer from pain at the back of the right thigh. At the end of three weeks this disappeared, and he was quite well for six or seven months, then the pain returned, and was severe for six weeks. Ten months before admission he had suffered from pain in the lower part of the back and down the posterior part of *each* leg. The pain continued for two months and then subsided; but it afterwards returned, and continued more or less until admission.

On admission he complained of of severe pain in the lower part of the back, down the back of each leg, and about the hips. Below the knee there was also pain in the anterior tibial regions. The pain was *worse at night*. There was no loss of power in the limbs, and the patient could walk quite well, but he stated that three weeks before admission he had great difficulty in rising from the sitting to the erect posture, and also when stooping he had the same difficulty in rising into the erect posture. He was only able to perform these movements by supporting the weight of his trunk by the hands placed on the knees or thighs. On examination. these movements could be performed quite well a few days after admission. The knee jerks were present, there was no affection of sensation to touch, pain, or temperature. The bladder and rectum were unaffected, the optic discs were normal. Under potassium iodide and mercurial inunction, considerable improvement occurred.

The following case, (recorded by Homén,) is a good example of syphilitic meningitis, producing symptoms similar to those in the first and third cases just reported. The diagnosis was verified by pathological examination.

Homén (6) reports a case of pachymeningitis of considerable interest. The patient was a man aged 45, who had been infected with syphilis at the age of 30. At the age of 41 he was rather suddenly attacked by severe pain in the lumbar region, which radiated especially to the left. This continued day and night for about seven weeks. About three weeks after the onset of the pain, dragging and stiffness of the left leg developed. Shortly afterwards the right leg was affected also. Walking became more and more difficult, and in two months after the onset of the pain, the legs were completely

paralysed. Bladder symptoms and a girdle sensation developed, and for a time a catheter had to be used. The pain in the back diminished; the legs remained paralysed for five weeks, but after antisyphilitic treatment there was gradual improvement, and the patient was able to walk with the aid of a stick. The bladder symptoms also diminished, but there was only slight diminution of the girdle sensation. The condition remained much the same during the next twelve months. Occasionally he felt pain in the legs, and the lumbar region of the spine became sensitive, especially to pressure.

Four years after the onset of his spinal symptoms he began to suffer from headache. At that time he was able to walk without the aid of a stick, but there was stiffness of the legs, especially in the morning. The headache increased; he began to suffer from vertigo and vomiting, and became dull mentally; but all the movements of the legs could be performed with considerable power. There was rigidity of the leg muscles, however, and the knee-jerks were much increased. Coma developed and death occurred.

Pathological examination revealed gummatous basal cerebral meningitis in the region of the pons and chiasma, and a gumma the size of a plum on the under surface of the cerebellum. In the lower dorsal region of the spinal cord there was most intense meningitis. From the 8th to the 11th dorsal nerve roots the cord was encircled by a thick layer of fibrous tissue, 7 to 8 mm. in thickness. Above and below the part mentioned the meninges and cord were normal to the naked eye. (See Fig. 1).

Microscopically at the level of the 9th and 10th dorsal nerves, sections of the cord and membranes showed that the spinal cord was surrounded closely by a thick layer of firm fibrous tissue, into which the membranes of the cord had been transformed. In places this fibrous layer was rich in nuclei. Posteriorly and on the right side of the cord, the nervous elements were for the most part destroyed and replaced by neuroglia, in the form of thick septa and connective tissue processes from the thickened meninges.

The adventitia of the arteries and veins, both in the cord and the surrounding meningeal fibrous layer, presented cell infiltration, and in some places the intima was also infiltrated. Ascending and descending secondary degeneration was found in the spinal cord at other levels.

The case is of interest from the fact that in the last stage of the disease, almost up to the death of the patient, the movements of the legs could be performed fairly well; and yet the pathological examination revealed a most intense pachymeningitis, which had invaded the cord posteriorly to a considerable extent.

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VI. MENINGO-MYELITIS.

It is admitted by almost all writers that the most common form of spinal syphilis is meningo-myelitis. As already mentioned (on page 11) in the last 32 cases of spinal syphilis which I have observed, 16 have been meningo-myelitis.

Sometimes the spinal affection is preceded by cerebral symptoms indicating a slight or temporary syphilitic affection of some part of the brain. In about a quarter of the cases which have come under my own observation one or more of the following slight or temporary cerebral symptoms have been present before the onset of the spinal affection :—Severe headache, often worse at night, vertigo, diplopia, slight external strabismus, left hemianopsia, temporary unilateral impairment of vision or hearing, slight paresis of one side of the face or tongue, slight numbness of the face, loss of speech for a very short time. Cases are on record in which other cerebral symptoms, such as vomiting, mental troubles, delirium, &c., preceded the spinal symptoms.

The symptoms of meningo-myelitis may be divided into two groups: (i.) The earlier symptoms due to changes in the meninges; (ii.) The later symptoms produced by extension of the changes to the spinal cord.

I. Early or Meningeal Symptoms.—The most prominent of these symptoms is pain in the back. The pain may be localised chiefly in the lumbar region; in some cases it is chiefly between the scapula, and in a few at the back of the neck, the difference in locality depending on the seat of the changes. According to Charcot and several

French authors, it is worse at night like the syphilitic headache. In many of the patients who have come under my own observation this has been a well-marked feature. In some cases there is spinal tenderness; and owing to compression or infiltration of the posterior nerve roots in the diseased meninges, there is frequently radiating pain in the parts to which the affected nerves are distributed. Thus, when the meninges of the lumbar and sacral parts of the cord are affected, there may be pain radiating along the course of the sciatic nerves, the anterior crurals, or along other nerve trunks of the leg. If the dorsal meninges are diseased, girdle pains are often produced. When the cervical meninges are affected, often the patient suffers from neuralgic pains along the course of the ulnar or other nerves of the arms. If the meninges of the upper cervical region are affected there may be radiating pains in the occipital region.

The nature of the pain varies; in some cases it is very severe, in others slight. Sometimes the neuralgic pains are present in the limbs, in various parts, for a short time; they then pass away, and other nerves are affected in an irregular manner.

When the anterior nerve roots are compressed motor symptoms are present. If the lesions should be limited to the dorsal region, these motor symptoms occur only in the intercostal or abdominal muscles, and as they do not produce any prominent symptoms they are not detected. If, however, the anterior roots of the cervical or lumbar nerves are compressed, then paralysis, with wasting of muscles, occurs in the arms or legs. Usually only single muscles or muscle groups are affected, and generally in one limb only at first. Thus I have seen the deltoid, biceps, and supinator longus paralysed and wasted; and at a later date all the muscles of one arm;

and then, finally, symptoms of a partially transverse lesion of the cord have developed. In this case probably the fifth and sixth cervical nerve roots were implicated on one side, and later the other motor nerve roots of the lower cervical region on the same side, before the cord was involved. Cases are on record in which the eighth cervical and the first dorsal nerve roots were involved first, before the cord was affected, and the symptoms of Klumpke paralysis produced (paralysis of the small muscles of the hand with anæsthesia following the distribution of the eighth cervical and the first dorsal nerves). In a case which I have recorded elsewhere, there was paralysis of the muscles supplied by one musculo-spiral nerve for several weeks before the onset of spinal symptoms. The affection of the arm muscles was probably due to a separate lesion, however.

The meningeal symptoms vary considerably in severity. They may be very prominent and cause the patient to seek medical advice for a long period before the onset of indications of involvement of the cord. In other cases they are slight; the patient only comes under observation when the cord has become involved, and then on careful inquiry into the history it is found that slight meningeal symptoms have preceded the affection of the cord.

The duration of the early meningeal symptoms, before the involvement of the cord also varies greatly—weeks, months, or even a year may elapse before the cord symptoms develop. In other cases the stage of meningeal symptoms is very short—a few days or weeks—and then the paralytic symptoms develop.

II. *Symptoms due to implication of the spinal cord.—Motor Symptoms.* The first indication of affection of the cord is often slight loss of power in the legs, which feel heavy in walking. The paresis gradually increases until

there is a well-marked paraplegia. In other cases paraplegia developes somewhat rapidly (sub-acute onset) and may become well-marked in a few days, whilst in a third group of cases paraplegia developes with great rapidity (quite suddenly or very acutely). This latter class of cases will be separately considered in the next section.

Sometimes there is a slight paresis for a long period and then complete paraplegia developes very suddenly. Occasionally there is one or more temporary attacks of paresis before the onset of severe paraplegia. In one of my cases there was temporary paresis of the leg, with retention of urine, several months after the syphilitic infection. Rapid recovered occurred, but a few weeks later the symptoms reappeared suddenly; the legs became completely paralysed, and remained so until death took place about nine years later. Sometimes symptoms of a unilateral lesion of the cord (hemiparaplegia or Brown-Séquard's paralysis) are observed at first; one leg is paralysed and the other leg anæsthetic, but as a rule these symptoms are only temporary, and both legs soon become paralysed and anæsthetic; though often the two sides are unequally affected for some time, the leg which is most paralysed being least anæsthetic. (Hemiparaplegia is more fully considered on page 100).

In a number of cases the bladder is markedly affected, and complete retention of urine may develop *before the legs are affected*, or when the legs are simply slightly weak and heavy. The bladder is occasionally completely paralysed for some hours, days or weeks before the legs are definitely affected. (In the chronic cases of Erb's syphilitic spinal paralysis a longer interval may elapse before the legs are affected. See page 76).

Attention has been drawn to this point by several writers, and it has been regarded as a sign of diagnostic

importance. In a number of the cases which have come under my observation, bladder symptoms have preceded the paraplegia, and I am inclined to regard it as a point in favour of syphilitic disease in any spinal case, but I do not think it is quite diagnostic, since I have occasionally obtained a similar history in other cases in which there was no evidence of previous syphilis.

The extent of the motor symptoms, when they have reached their greatest development, varies considerably. There may be complete paralysis of both legs. In many cases, however, the paralysis never becomes complete. Sometimes the legs are unequally affected, and when both are finally paralysed, as just mentioned, one is often affected some, hours, days or weeks before the other. When the upper part of the dorsal region is affected the intercostals are paralysed, the extent varying with the level of the lesion. If the lesion is in the dorsal region (and this is most frequently the case) the legs frequently become spastic in course of time: there is then rigidity of the limbs to passive moments. The knee jerks become increased, ankleclonus developes; the skin reflexes may be increased or diminished. Sometimes the knee jerks are increased, but ankle-clonus is absent. Occasionally the knee jerks are absent and the legs flaccid when the lesion is in the dorsal region. If the changes are chiefly in the lumbar enlargement, then the legs become wasted and symptoms of atrophic paralysis develop, and the knee jerks and ankle-clonus are absent. If the changes are in the cervical region, then atrophic paralysis of the arms, with spastic paralysis of the legs, may develop. In the majority of cases, however, the arms are not affected, because the lesion is in the dorsal region of the cord.

On electrical examination, usually no changes are found (except slight increase or diminution of excitability); but

if the anterior horns are affected in the cervical or lumbar regions, then the muscles supplied from these parts undergo marked atrophy, and the reaction of degeneration may be obtained on electrical examination.

When the patient's legs are not completely paralysed, if he should be able to walk, the gait is usually spastic. Occasionally there is ataxia, and Rhomberg's symptoms may be present.

Bladder symptoms.—If the bladder is not affected before the legs, the onset of paraplegia is often followed by marked paralysis of the bladder and rectum.

Frequently there is retention of urine followed by dribbling from an over-distended bladder, or there may be spasmodic or paralytic incontinence of urine and fæces. Even when the loss of power in the legs has been very slight, I have seen the bladder symptoms very pronounced. As already mentioned, often retention of the urine occurs before the legs are affected.

Usually the bladder symptoms are followed by cystitis. The urine becomes alkaline, then ammoniacal; a deposit of pus and phosphates occurs. Numerous bacteria are present, and sometimes abundance of ammonium urates. In some cases hæmaturia occurs. This may be owing to the intensity of the cystitis (sometimes it is due to the passage of the catheter). Urethritis may also develop, and a collection of pus is often found at the anterior urinary meatus. Balanitis or œdema of the prepuce often occurs at a late stage. The cystitis is frequently followed by pyelo-nephritis and pyæmia.

Sensory Symptoms.—Sensation is usually affected, but not so markedly as motion. Paræsthesia is common, the patient complaining of numbness or tingling in the legs. Anæsthesia in some form is usually present. Occasionally it is well marked and complete, but frequently

it is incomplete, and very often slight. Occasionally there is anæsthesia to all forms of sensation in the leg and lower part of the trunk. More frequently there is a partial or incomplete anæsthesia. There may be slight diminution to all forms of sensation. In other cases tactile sensation is normal, or only slightly affected, whilst there is marked affection, or complete loss, of sensation to pain and temperature.

In a number of cases I have found the *sense of temperature* affected, whilst other forms of sensation have been normal; and in other cases I have found marked loss of temperature sensation, whilst the sensation of pain has been only slightly affected, and tactile sensation has remained normal.

When the temperature sense is affected, the patient may be unable to recognise the difference between hot and cold test tubes. I have also observed cases in which the thermo-anæsthesia has been for cold objects only. Warm objects have been readily recognised as such, but cold objects have felt warm to the patient; and this loss of sensation for cold has been the only form of anæsthesia to be detected. *In all cases of spinal syphilis, before concluding that sensation is unaffected, it is important to examine the sense of temperature very carefully—especially the sensation for cold.* Further, it is also necessary to examine the gluteal region and back of the thighs, as well as the front of the thighs and legs. In one case (under the care of Dr. Dreschfeld) I found the only form of anæsthesia was loss of sensation to cold, on the gluteal region, and along the back of the left thigh, the outer side of the leg (below the knee), and the foot—corresponding to the distribution of the sacral plexus.

The anæsthesia of spinal syphilis is distributed in the same manner as in non-specific spinal lesions. When

well marked, it may extend up to the umbilicus, to the epigastrium, to the lower intercostal spaces, or higher, according to the situation of the lesion. As mentioned already, occasionally there is a unilateral anæsthesia—loss of sensation on one leg and one side of the abdomen (or abdomen and part of the chest.) All forms of sensation may be lost in this unilateral anæsthesia, or there may be unilateral loss of sensation for pain and temperature only. In these cases there is usually paralysis of one leg only (the leg which is not anæsthetic), or if both legs are paralysed, one leg is affected much more than the other. (See page 100).

Bed sores are very liable to develop over the sacral and gluteal regions. They may become very extensive and deep, and by septic-absorption from the sloughing surface, pyæmia may occur.

Loss or diminution of sexual power may be noted. Erection of the penis may be slight or incomplete: the sexual act may be more or less imperfect, or sexual power may be completely lost. This is occasionally one of the first symptoms of the disease. (See also p. 102).

It remains to refer to several points in the symptomatology which have been regarded as characteristic by various authors, *i.e.*, the association of meningeal and root symptoms with those pointing to involvement of the cord; the multiplicity and partial or incomplete nature of the symptoms, as well as their variability. When the patient is frequently examined, it is often found that the extent of the paralysis or paresis varies from time to time. The anæsthesia and bladder symptoms may also vary in the same way. The knee jerks at one time may be present, a little later absent, whilst afterwards they may return.

Abortive Cases.—I have seen some cases in which after

the patient has suffered from pain in the back for a short time, retention of urine has occurred, and this has been followed by slight loss of power in the legs ; but in a very short time all the symptoms have disappeared.

Termination :—

I. *Recovery*. Some cases recover in course of time. Occasionally rapid and complete recovery occurs when the symptoms have not been very severe ; but often the recovery is not quite complete, some trace of the affection being left behind.

II. The symptoms may *remain stationary* for many years, and death may occur from some inter-current or accidental affection, such as bronchitis, broncho-or croupous pneumonia, phthisis, &c.

III. In some cases the symptoms present marked *fluctuations* from time to time, without any permanent recovery taking place.

IV. *Death* may occur even at an early, or comparatively early date, as a result of the spinal affection, directly or indirectly. The most common cause of death is cystitis, followed by pyelo-nephritis and septic poisoning. Another common cause of fatal termination in septic absorption from a large bed sore. Many cases die from lung complications, especially broncho-pneumonia or basal pneumonia, and often this is caused by paralysis of the lower intercostals. Death may also be caused by the lesion extending upward, and producing paralysis of both intercostals and diaphragm, and so giving rise to asphyxia.

Diagnosis.—The general remarks made in section III, on the diagnosis of spinal syphilis, apply especially to the most common variety—meningo-myelitis.

It remains only to briefly mention a few diseases which

are most likely to be mistaken for syphilitic meningo-myelitis.

Spinal caries gives rise to pain in the back, and signs of irritation of nerve roots, along with paraplegia. But the root and meningeal symptoms are usually localised to one limited region of the cord—corresponding to one or two vertebræ—whilst in spinal syphilis they are more diffuse. Also there are usually the characteristic "bone symptoms" in caries, prominent vertebral spines, &c. (See p. 29).

In every case, of course, before giving a diagnosis of spinal syphilis, the spine ought to be carefully examined for signs of caries.

The meningeal symptoms (pain, &c.) usually serve to distinguish the cases from "*idiopathic*" *myelitis*. The points referred to in section III. are of importance in the diagnosis between specific and non-specific *meningo-myelitis;* and also in the diagnosis between syphilitic meningo-myelitis and *spinal tumour*.

The presence of pain, bladder symptoms, and anæsthesia separates syphilitic meningo-myelitis from *amyotrophic lateral sclerosis*.

The actual paresis or paralysis is sufficient to prevent confusion with *locomotor ataxia* in most cases; but some cases of spinal syphilis simulate locomotor ataxia. The differential diagnosis is considered on page 111.

The remarks on page 104 will apply to the differential diagnosis between *disseminated sclerosis* and syphilitic meningo-myelitis, and those on page 108 to the differential diagnosis of the latter affection from *syringo-myelia*.

In *ataxic paraplegia*, the late occurrence of bladder symptoms and anæsthesia, (if these occur at all), and the absence of pain, are usually diagnostic. The other points in section III. are also of importance.

Pathological Anatomy of Meningo-Myelitis.

Pathological observations, made during recent years, have shown the importance of vascular changes in the production of spinal syphilis; they are very frequently present, and, no doubt, they often form the starting point of the pathological changes in the cord. Degeneration or softening follows the impairment of blood supply, caused by the vascular disease, and, finally, in the degenerated region, sclerotic changes occur. In some cases, however, no definite characteristic vascular changes have been found, or, if present, the vascular changes have been very slight. It is possible that sometimes these have been instances of the accidental occurrence of a non-specific spinal affection in an individual who has happened to have suffered from syphilis at a previous date.

The vascular changes are, however, so frequent and important as to merit a description first. They are similar to those met with in syphilitic affections of other organs. The small vessels in the meninges and periphery of the cord, are most affected.

Arteries.—The walls of the spinal arteries often present a well-marked thickening, which causes the lumen of the vessel to be diminished, and in some cases obliterated. Often the adventitia of the arteries is most affected, and is infiltrated with round cells; in other cases the chief change is a proliferation of the endothelium of the intima —endarteritis. Often both internal and external coats are affected, the changes being sometimes most marked in the adventitia, sometimes in the intima. The middle coat is least often affected, and never shows such marked changes as the other two coats.

Whether the arterial affection begins in the intima or

adventitia is a disputed point. Heubner regards the first view as the more probable. Baumgarten, Friedländer and others, the second. Apparently it commences sometimes in the one, sometimes in the other.

At a comparatively early stage the thickened intima consists of cells to the inner side of the elastic lamina—partly small round cells, partly larger spindle-formed or angular cells. The adventitia is infiltrated with round cells. The median coat is free, or only slightly affected.

The thickening of the coats may be so great that the lumen is almost, or completely obliterated—arteritis syphilitica obliterans.

Fig. 2.

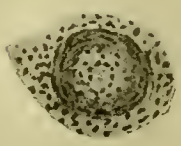

Fig. 3.

Figs. 2 and 3.—Small meningeal arteries from a case of syphilitic meningo-myelitis. Endarteritis and periarteritis. Logwood and Eosin (Zeiss, D.)

At a later period the cell infiltration passes on to the formation of fibrous tissue. The arterial coats are then more fibrous, and contain less cell elements. The middle coat is unaffected or only atrophied in parts. In old-standing cases the wall of the small vessels may contain a broad layer of homogenous or "hyaline" material.

Though there is nothing actually characterastic in these changes; still, in non-specific arteritis, leading to sclerosis, there is not the tendency to such a marked cell proliferation in the coats, especially in the adventitia (Ziegler).

The vascular changes are probably produced by the action of some toxin. Whether they start in the intima

or the adventitia (and probably the latter is more frequently the starting point than the former), the changes in the intima are the most important, since they lead to narrowing of the lumen or occulsion of the artery: also in the diseased vessels thrombosis may occur.

Sometimes the infiltration of the vessel walls forms a localised patch, or a miliary gumma (but this is more common in the veins).

As a result of the vascular disease, occasionally thinness of the vessel wall, and a minute aneurism may develop.

The *veins* are affected in a similar manner to the arteries. In many cases the veins are more affected than the arteries, and this has been especially the case when the post mortem examination has been made at an early date after the syphilitic infection (Greiff, Siemerling, Lancereaux, Lamy, Sottas).

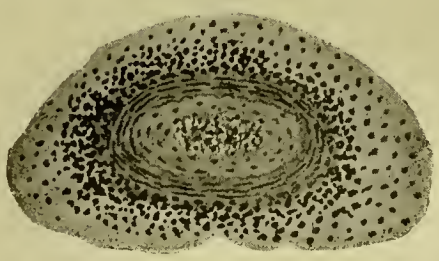

Fig. 4.

Meningeal vein from a case of syphilitis meningo-myelitis. Endo- and peri-phlebitis Small thrombus in the centre. Logwood and Eosin (Zeiss, D.)

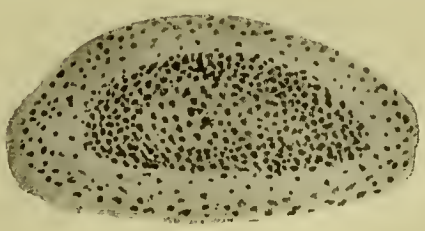

Fig. 5.

Meningeal vein complete by obliterated endo-phlebitis obliterans. Logwood and Eosin (Zeiss, D.) Case of syphilitic meningo-myelitis.

Often there have been marked changes in the veins whilst the arteries have been almost unaffected ; in other cases, however, the changes in the veins have been much less than the arterial changes.

The disease affects first the external coats of the veins ; but soon all the coats are involved, and the wall may be so much infiltrated and thickened that the lumen is obliterated — phlebitis obliterans. Sometimes miliary gummata develop in the walls of the veins. Within the cord the small vessels often present a marked hyaline thickening of their walls.

The vascular changes occur both in the membranes and in the cord ; their severity varies, even in the same case, at different parts of the cord, and at different parts of the same vessel ; and the vascular changes and the degeneration of nerve elements do not always run parallel.

The *meninges* present marked vascular changes, especially near the posterior nerve roots. In some cases, besides the vascular changes, there is simply a zone of round cells (leucocytes) just around the vessels, whilst a short distance from vessels the meninges appear normal. In more advanced cases there is a diffuse infiltration of round cells everywhere in the meninges, though this is most marked just around the vessels. Sometimes there is a gummatous infiltration of the meninges with much thickening. In old standing cases the meninges present fibrous thickening. In some cases the meningeal changes are extensive and the cord changes slight ; in other cases the meningeal changes are very slight and the cord changes marked.

Changes in the Cord.—The structure of the spinal cord may be damaged by (1) cell infiltration or gummatous infiltration ; (2) by softening or degeneration, owing to obliteration (complete or partial) of the nourishing vessels.

In some parts the changes are only in the periphery,

whilst in other parts they extend over a great portion of the transverse section of the cord.

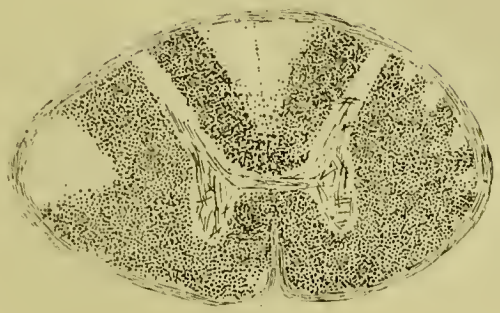

Fig. 6.

Syphilitic meningo-myelitis. Weigert's stain. Wedge-shape mass of gummatous infiltration in the R lateral column. Meninges thickened.

From the thickened or inflamed pia-mater broad septa of cell infiltration, or of fibrous tissue, may extend into the white matter; or wedge-shaped masses of granulation tissue may invade the cord from the periphery. These wedge-shaped patches may consist chiefly of round cells; or they may have undergone degenerative or caseous change in the centre and fibrous changes at the periphery, so as to present a gummatous appearance. Also minute miliary gummata are sometimes detected in the cord, in the walls of the vessels, and in the meninges. Occasionally a large gumma may invade the cord (see p. 94). Patches of softening, similar to cerebral softening, may also be found, and occasionally there are small hæmorrhages.

The nerve fibres are degenerated in the parts most affected. They are broken down into granules and fragments. Leucocytes and compound granular cells are found in the affected area and in the perivascular lymphatic spaces.

The grey matter is usually less affected than the white, especially in cases in which the symptoms develop slowly.

But when the gummatous infiltration, or the softening, extends to the grey matter of the anterior horns, the nerve cells and fibres in this region become degenerated also.

The vessels within the cord are very frequently affected, and in many cases, as already mentioned, these vascular changes appear to be the starting-point of the destruction of nerve elements. As described already, the arteries may be much thickened, and the lumen diminished or obliterated. Endarteritis is the chief change in some cases; in others cell infiltration of the adventitia is the prominent feature.

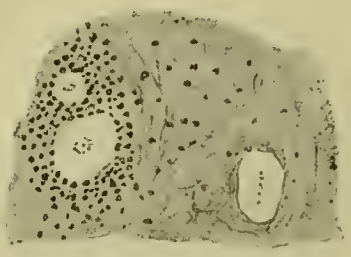

Fig. 7.

Hyaline vessels from case of syphilitic meningo-myelitis. Vessels to the right surrounded by cell infiltration; that to the left by sclerosis. Logwood and Eosin (Zeiss, D.)

The veins may present endo-phlebitis, peri-phlebitis, or obliterating phlebitis. The smallest vessels often present marked hyaline thickening of their walls when the disease is of long standing. These hyaline vessels are often surrounded by cell infiltration. In the early stage the small vessels are often dilated and full of blood, and surrounded by cell infiltration. Capillary hæmorrhages are sometimes present in the softened parts.

Nerve Roots.—The disease of the meninges often affects the nerve roots, and by pressure or cell infiltration atrophy of the nerve fibres is produced. The degeneration of the roots is most intense where the meningeal changes are most marked.

Secondary Sclerotic Changes.—The degenerative changes and softening in the cord are followed by the development of new firm fibrous tissue, and sclerosis is produced in the affected parts if the patient should live long enough. The vessels become hyaline: they are surrounded by fibrous tissue, and their walls much thickened.

Secondary ascending and descending degeneration may be found at a late period, and the localisation of the degenerated tracts is the same as in non-specific cases.

The softening in the cord occasionally leads to the formation of cavities. (See p. 108).

In meningo-myelitis the changes are most irregular in their distribution. The meningeal affection may be very diffuse, but it reaches its greatest intensity in the dorsal region. The cord affection is usually most marked in the dorsal region also.

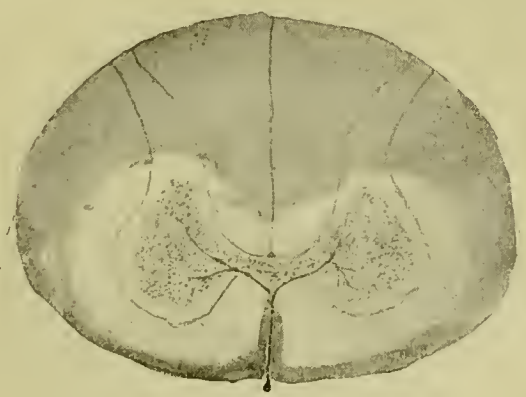

Fig. 8.

Diagram showing division of transverse section of spinal cord into three zones. (i.) Central zone (dotted) in the centre of the grey matter, which is supplied by the anterior median or central arteries of the cord. (ii.) A peripheral zone (shaded) supplied by the peripheral arteries of the cord. (iii.) A third zone (pale) supplied by both peripheral and central arteries.

The changes are most intense at the periphery of the transverse section of the cord.

The observations of Kadyi and others, have shown that the transverse section of the cord may be divided into three zones according to the blood supply (see Fig. 8): (i.) A central zone (dotted in the diagram), occupying the centre of the grey matter, which is supplied by the anterior median or central arteries of the cord. (ii.) A peripheral zone (shaded in the diagram) supplied by the peripheral arterial system of the cord. (iii.) A third zone (pale) in the diagram, which is supplied with vessels from both systems.* This zone contains the outer part of the grey matter and the adjacent white matter.

Now, in spinal syphilis, this third intermediate zone is usually least affected. The peripheral zone is most diseased, and it is chiefly the part supplied by the posterior and lateral arteries which is affected. (See Fig. 9). Probably in a few cases the changes are localised to the central region (see p. 106), owing to disease of the anterior median arteries.

Fig. 9.

Section showing distribution of anterior and posterior arterial system of the cord. Part shaded by dots = area supplied by posterior arteries of the cord.

* "The relation of diseases of the spinal cord to the distribution and lesions of the spinal blood vessels;" by R. T. Williamson. Lond., 1895.

VII. ACUTE SYPHILITIC PARAPLEGIA.

("Acute Syphilitic Myelitis.")

Sometimes the symptoms of spinal syphilis resemble those of acute myelitis, or spinal hæmorrhage. There is a sudden onset of paralysis of both legs, with paralysis of the bladder and rectum (retention of urine, or retention with dribbling, and incontinance of fæces). The lower intercostals may be paralysed, sometimes all the intercostals, and, occasionally, but rarely, the arms are affected also. Cystitis and bed-sores develop, and sensation is usually affected. There may be anæsthesia to all forms of sensation, or analgesia and thermo-anæsthesia whilst tactile sensation is preserved, or thermo-anæsthesia may be present and other forms of sensation normal. Frequently the sensory disturbances are slight, whilst the motor are very well marked. The knee jerks may be present or absent. In the cases which have come under my observation recently, ankle-clonus has been absent.

The acute cases referred to in this section are those in which parylasis of the legs becomes complete in the course of a few hours, or in a day or two.

Prodromal symptoms are often present; these may be spinal or cerebral. The following spinal prodromal symptoms may be noted: Pains in the back; pains in the legs; paræsthesia in the legs; girdle sensations; tiredness of the legs; bladder symptoms (retention of urine). There are often remissions and exacerbations in these symptoms. Prodromal symptoms may also occur in non-specific myelitis, but Rosin points out: (1) That the prodromal symptoms develop for a longer period

before the onset of the paraplegia in syphilitic cases than in non-specific cases. (2) That in specific cases there are variations in the intensity of these symptoms, and they may be present in a part which is not subsequently involved in the paralysis; also in cases which afterwards present the symptoms of a pure spinal affection, prodromal *cerebral* symptoms may be present. (3) Retention of urine often occurs, as a prodromal symptom, for some days or weeks before the development of paraplegia in syphilitic cases, whilst in non-syphilitic myelitis this is rare. (4) Variations in the condition of the patella reflex may occur.

As regards the third point in the cases which have come under my own observation, retention of urine has often been a prodromal symptom in syphilitic cases, whilst in non-syphilitic cases this has been very rare. Still I do not think the point is diagnostic, since in a few cases of paraplegia (? acute myelitis), in which I could obtain no history or indication of previous syphilis, there has been retention of urine for some time before the onset of the paraplegia.

The prodromal cerebral symptoms are chiefly headache, vertigo, noises in the ears (and others mentioned in section VI).

Often acute syphilitic paraplegia or myelitis has occurred at a comparatively early period after the syphilitic infection. Cases have been recorded which have occurred six or seven months after the primary syphilis: in other cases the paraplegia has developed a few years or many years after infection. So that this form of spinal syphilis may develop any time between six months and ten years or more after the infection; but in seven out of twelve cases collected by Rosin, the disease occurred during the first three years.

In a few cases the paraplegia develops with great rapidity, it follows a short and insignificant prodromal period and terminates fatally in a few days or a few weeks. In such cases lesions may be found in the cord, which show clearly the syphilitic nature of the disease.

Analysis of Cases.

The following are points of interest with respect to six cases of acute syphilitic paraplegia which I have recently had the opportunity of following carefully.

All the six patients were males.

The ages were 28, 28, 32, 35, 38, 43 years respectively.

In these cases the dates of onset after the syphilitic infection were as follows: In one case 7 months only, in the others, 2 years, 3 years, 5 years, ? 19 years, ? 25 years respectively.

Onset and Prodromal Symptoms.—In one case the onset was almost as sudden as in spinal hæmorrhage. The patient (who was under the care of Dr. Bury), had suffered from a severe pain in the lumbar region for one hour in the morning, this disappeared, and he followed his employment all day. About 10 p.m. he sat down for a quarter of an hour. On attempting to get up, he found that both legs were paralysed. There was retention of urine from the same evening. A distinct syphilitic rash was present at the onset of the paralysis.

In three cases the first symptom was pain in the back, which continued more or less until the onset of paralytic symptoms. In two of these cases the paralysis occurred four days after the onset of the pain (in the one case paraplegia and retention of urine came on at the same time; in the other there was retention of the urine first and paraplegia next day). In a fourth case there was pain in the back more or less for one month, then retention of

urine, and next day paraplegia. (Fully recorded on page 58). In a fifth case the symptoms commenced with retention of urine and unsteadiness in walking; paraplegia became complete next day. In the sixth case there was retention of urine first, very soon afterwards one leg was paralysed, and next day both legs were paralysed.

The early implication of the bladder is interesting. In three of the cases there was retention of urine, even a short time before the development of paraplegia. In the other three the symptom appeared along with the paraplegia.

Motor Symptoms.—In all six cases there was complete paralysis of both legs, but the arms were not affected.

In all six cases the paralysed limbs were flaccid.

In three cases the knee jerks were absent, and there was no ankle-clonus. In one case the knee jerks were absent at first for several weeks, but afterwards returned. In two cases the knee jerks were present, but ankle-clonus absent.

In all of the six cases ankle-clonus was absent.

Bladder.—In all of the cases there was retention of urine, or retention of urine with dribbling; and in all cystitis developed, and was followed by symptoms of septic absorbtion, irregular pyrexia and rigors. In five of the six cases large bed sores developed.

Sensation.—In two of the cases there was loss of sensation to tactile, and painful impressions on the legs, abdomen, and lower part of the thorax. In one case there was impairment of sensation on the legs and abdomen, but not complete anæsthesia. In two cases there was analgesia, with thermo-anæsthesia on the legs and abdomen, but bactile sensation was not affected: in one

of these two cases, at a later date, there was thermo-anæsthesia only.

In one case (which terminated fatally in two months), the head and the point of a pin could be distinguished and localised on the legs and abdomen, though there was complete paralysis of both legs, retention of urine, and a large bed-sore.

Termination.—The cases of "acute syphilitic paraplegia" often terminate fatally at an early date. The cause of death is frequently a pyæmic condition following cystitis and consequent pyelo-nephritis, or following septic absorption from a large bed sore. Another frequent cause of death is basal pneumonia, and often in such cases there has been paralysis of the lower intercostals. Sometimes death has been due to asphyxia from paralysis of all of the intercostals, and afterwards of the diaphragm.

Five out of the six cases mentioned above, terminated fatally.

In one case, death occurred in 15 days (see page 58) from the onset of paraplegia, the cause of the fatal termination being cystisis and pyelo-nephritis.

In another case death occurred in 20 days from basal pneumonia, cystitis and pyelo-nephritis.

A third case terminated fatally in two months, from left-sided basal pneumonia and cystitis and pyelo-nephritis.

A fourth died from cystitis and pyelo-nephritis in five months.

A fifth case terminated fatally in about 12 months. Cause of death unknown (? cystitis and pyelo-nephritis).

The sixth case recovered completely, though at first, there was marked paraplegia and retention of urine. Cystitis developed and became very severe, but in time it subsided. The patient was following his employment as

a policeman 18 months after the onset of the paraplegia.

These very acute cases often run a fatal course in spite of vigorous anti-syphilitic treatment.

The *prognosis* is very grave. Many cases terminate fatally in a very short time ; in others, the lesion remains stationary for a time, or shows slight improvement, and then death occurs from complications. The causes of death have been already indicated In a few cases partial or, occasionally, complete recovery occurs.

In the differential *diagnosis* of acute paraplegia, the general indications given in section III. are usually sufficient to enable a correct conclusion to be arrived at.

Of course, in every case, the question of vertebral caries must be considered, and the back examined for prominent vertebral spines, tenderness on percussions and movement, &c. Evidence of tuberculosis elsewhere are of importance.

Usually all other affections, except idiopathic myelitis, can be excluded readily, and the points already mentioned are sufficient to enable a decision to be arrived at.

Pathology of acute syphilitic paraplegia.—From the frequency and importance of cerebral syphilitic thrombosis one would expect that thrombosis in the spinal vessels would also play an important part in the pathology of spinal syphilis of acute onset—so called "acute syphilitic myelitis;" but as yet the pathological evidence on this point is small. Hence the pathological changes in the case described below are of considerable interest. I am indebted to the kindness of Dr. Steell for the opportunity of making the pathological examination.

*Spinal Thrombosis and Hæmorrhage due to Syphilitic Disease of the Vessels.**—The patient was a man aged twenty-eight, who was admitted under the care of Dr. Graham Steell at the Manchester Royal Infirmary on

* I have recorded this case in full, *Lancet*, July 7th, 1894.

PLATE I.

Unstained sections of spinal cord, showing position of hæmorrhage (brownish red) in case of acute syphilitic paraplegia (recorded page 58).

Sept. 22nd, 1893. About one month previous to admission the patient began to suffer from pains in the back, under the left scapula. There was sometimes pain under the right scapula; sometimes pain extended around the left axillary region, and as far forwards as the cardiac region. The pain was not severe, but continued more or less until the onset of paralysis. On Sept. 20th he first noticed some difficulty in passing his urine, but at that time he could walk quite well and had no pain. In the evening his gait was unsteady. Early in the night, after going to bed, he felt a desire to micturate. He got up, but found that he was quite unable to pass his urine. He was able to walk fairly well, and went downstairs for a short time. He then returned to bed and fell asleep. On awakening in the morning he found that both legs were paralysed. He could just "put the legs together," but could not stand. He stated that there was loss of feeling in the legs. There was no pain anywhere and no girdle sensation. There had been loss of control over the rectum and retention of urine ever since the night of Sept. 20th. A catheter had been passed twice daily. There was no history of injury to the back, but there was a history of alcoholism and syphilis. Two years previously he had had a chancre followed by sore-throat and falling off of the hair, for which he was treated by a medical man with iodide of potassium.

On Sept. 25th both legs were completely paralysed; the limbs were flaccid. Knee-jerks, ankle clonus, and cremasteric, abdominal, and epigastric reflexes were absent on both sides. Very slight plantar reflexes were obtained. There was no paralysis of the upper limbs, intercostal muscles or diaphragm. There was loss of sensation to tactile and painful impressions (to the head and point of a pin) on the legs and trunk as high as the sixth intercostal space. In this interspace there was impaired sensation, but in the fifth intercostal space, and in all parts above, the sensation to tactile and painful impressions was good. There was no band of hyperæsthesia, no girdle sensation, and no spinal curvature. There was complete loss of control over the rectum and retention of urine. The rate of respiration was 36 per minute. There was a large amount of expectoration, and submucous râles and rhonchi were heard all over the chest. Nothing of importance was detected in the other organs. The urine contained pus and a large amount of blood.

On Oct. 3rd anæsthesia (to the head and point of the pin) extended as high as the sixth rib. There was now a zone of hyperæsthesia in the fifth interspace. The knee-jerks were absent. The urine contained blood and pus. The mental condition was very dull.

On the 4th there was profuse diarrhœa and abundant expectoration. After admission rigors occurred once or twice daily, and the temperature was remittent or intermittent, high in the evening (on two occasions reach-

ing 103° F.). lower or normal in the morning. A bedsore developed on the right buttock. The motions were passed involuntarily, and the retention of urine continued. The area of anæsthesia remained unaltered. The patient continued in much the same condition until his death on the 5th. Death appeared to be due to the pyæmic condition.

Necropsy (abstract).—Over the sacrum and right gluteal region were bedsores, and an old cicatrix was found on the glans penis. The brain was congested. The lateral ventricles contained a considerable amount of clear serous fluid. There were no signs of tubercle, syphilis, or other gross lesion. The lungs were congested and œdematous, but crepitant in all parts. The heart weighed 11oz., and the valves were normal. The peritoneum was normal; the gastro-intestinal tract was congested. The liver weighed 4lb. 4oz., and was slightly enlarged. The spleen was enlarged slightly and weighed 10oz. The kidneys presented well-marked signs of pyelo-nephritis. The ureters were acutely inflamed and there was very well-marked cystitis. The spinal cord showed no evidences of softening at any part, and was very firm and in exceedingly good condition. The meninges were congested, but no other naked-eye changes were detected on the surface of the cord. On section, about the middle dorsal region a hæmorrhage was found, occupying the whole of the grey matter of the cord on the left side. Scrapings from the cut surface of this part were examined in a 1 per cent. solution of osmic acid at the time of the necropsy, but no compound granular cells could be detected microscopically. The cord was not softened in the region of the hæmorrhage. No attempt was made to determine the limits of the lesion at the necropsy; the cord was placed to harden in Müller's fluid. After hardening, a number of transverse divisions were made in the dorsel region, and on naked-eye examination a large hæmorrhage dark brownish-red in colour, was found in the grey matter of the left side of the cord, about the mid-dorsal region. The vertical extent was about one inch and three-quarters (downwards from the mid-dorsal region). At both the upper and lower parts the hæmorrhage was limited to a small streak in the posterior horn (left). About the centre of the lesion the hæmorrhage occupied the whole of the grey matter of the left side, and extended across the grey commissure into the central part of the grey matter of the right side. For one inch below the inferior part of the hæmorrhage a few white points were seen scattered through the white matter of both sides; also a few white streaks were seen in the white matter in some sections. The same condition was met with for about two inches above the highest part of the hæmorrhage. In the cervical and upper dorsal regions (above the lesion) the posterior columns presented the naked-eye appearances of ascending degeneration—*i.e.*, these parts were paler than the rest of the white matter. To the naked eye there did not appear to be

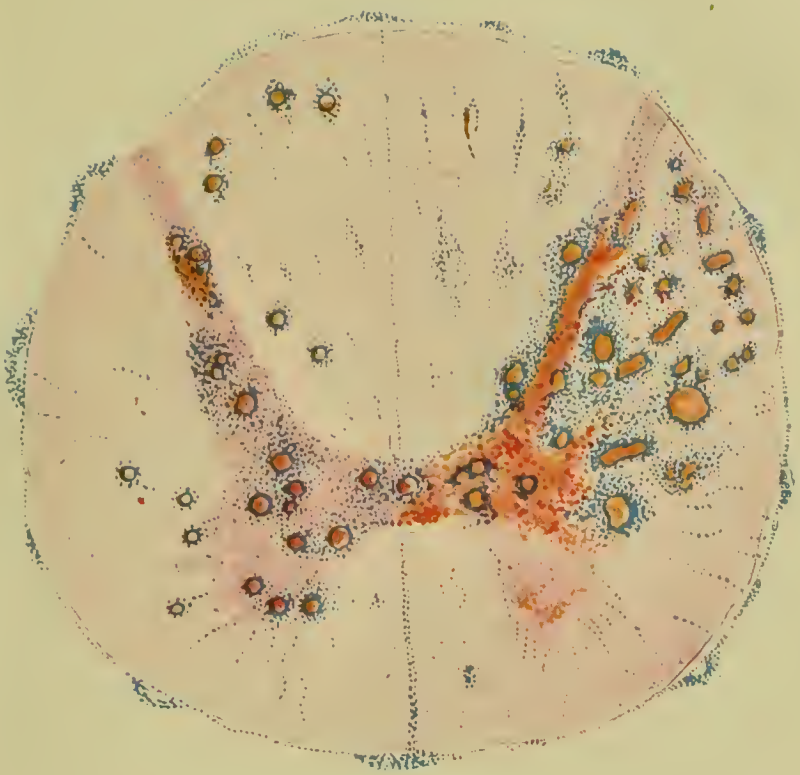

PLATE II.

Spinal thrombosis and hæmorrhage due to syphilitic disease of blood vessels. Very low power. Logwood and Eosin. (Hæmorrhage and dilated vessels coloured brown).

any descending degeneration below the lesion. The whole of the affected part of the cord, (*i.e.*, at the seat of hæmorrhage), was cut into sections (celloidin method), and every third examined. Altogether some hundreds of sections were obtained. Numerous sections were also taken a little above and a little below the seat of hæmorrhage, and in the cervical and lumbar regions. The sections were stained with logwood, aniline blue black, logwood and eosin, and according to Weigert's method.

Microscopical examination showed that in the parts which, to the naked eye, appeared dark red (hæmorrhagic), the tissues were infiltrated and replaced by a mass of red blood corpuscles. At the highest part of the lesion, the hæmorrhage was limited to a small streak in the left posterior horn of grey matter; at a lower level it extended to the outer part of the grey matter (first Fig. in plate 1); a little lower it became more extensive, occupied the whole of the grey matter, and spread in irregular patches to the white matter of the left half of the cord (second Fig. in plate 1). Near the middle of the vertical extent of the lesion the hæmorrhage extended to the grey commissure, and then over to the grey matter of the right half of the cord, but never became so extensive in the right half of the cord as in the left, and occupied only the centre of the right grey matter (third Fig. in plate 1). At a lower level the hæmorrhage was confined to the grey matter of the left side (fourth Fig. in plate 1), and at the lowest part only a small streak of hæmorrhage was seen in the posterior horn. A few very small patches of hæmorrhage, chiefly around dilated vessels, were scattered irregularly in the white matter. Next to the hæmorrhages the most striking feature of the sections of the cord was the great number of enormously dilated bloodvessels, especially in the grey matter and the adjacent white matter of the left side at the seat of the hæmorrhage. Some of these vessels were equal in diameter to the breadth of the grey commissure. Dilated vessels were seen in the grey matter for a short distance above and below the hæmorrhage and also in the grey matter of the right side of the cord; but the most marked dilatation was seen on the left side in the region of the hæmorrhage. At one point, however, where the hæmorrhage was greatest—*i.e.*, where it occupied almost the whole of the grey matter of the left side—only a few of these dilated vessels were seen. The vessels were packed full of red corpuscles, and very many were thrombosed. In the interior of these thrombosed vessels close to the vessel wall, was an irregular layer of granular-looking substance (stained pink with eosin), containing many fine fibres, nuclei, and nucleated cells, whilst red blood corpuscles, with leucocytes, were seen nearer the centre. In some of the vessels this new-formed tissue occupied more than half of the lumen, and extended in an irregular manner to the centre (see plate III.) The walls of these vessels were thickly covered with nuclei, and there was well-marked

perivascular cell infiltration, the amount varying at different parts. A large number of these thrombosed vessels were surrounded by hæmorrhage. In a good number of sections, the anterior median artery was obstructed by a thrombus. In a few sections, the central canal was surrounded by a zone of tissue crowded with nuclei; in others it was surrounded by hæmorrhage. Scattered irregularly in the white matter, in some of the sections, a few small patches were seen in which the interstitial tissue was a little in excess and its nuclei increased in number and size. As already mentioned, for two inches above and about one inch below the hæmorrhage, a few irregularly distributed white points and streaks were seen on the cut surface of the hardened cord, and microscopical examination showed that these were due to dilated vessels with marked perivascular cell infiltration. At no part of the cord were any compound granular cells to be seen; and the inflammatory changes—excess of nuclei and cell infiltration—were always just around the vessels. In all parts, even the upper cervical and lumbar regions, there were distinct changes in the meninges. These consisted of infiltration with round cells and marked vascular changes. The cell infiltration was only slight in the cervical and lumbar regions. The vessels of the meninges and of the cord showed distinct syphilitic changes; they were best marked, however, in the meninges. The walls of many vessels were greatly thickened. In many there was well-marked endarteritis—sometimes so extensive that the lumen was almost obliterated—and in a few arteries no lumen could be detected. Some of the arteries were obstructed by a thrombus. The adventitia was greatly thickened and infiltrated with round cells; there was also perivascular cell infiltration. Similar changes were met with in many of the veins—endophlebitis, periphlebitis, thrombosis. (Figs. 2, 3, 4, and 5).

Pathological diagnosis.—Syphilitic disease of vessels (endarteritis, periarteritis, endophlebitis, periphlebitis); thrombosis; hæmatomyelia; leptomeningitis.

The onset of the symptoms in the case recorded above was not quite so sudden as is usual in so-called primary spinal hæmorrhage. The slight pain in the back for a month previous to the paralysis, the retention of urine, and the slight unsteadiness in gait for some hours previous to the actual paraplegia, together with the history of syphilis, appeared, during life, to be points against a primary hæmorrhage, and more in favour of a diagnosis of very acute myelitis. But the microscopical examination showed clearly that the primary change was syphilitic disease of the vessels, and that this had been followed by thrombosis and hæmorrhage. There can be no doubt that the thrombosis of vessels occurred before the hæmorrhage and not as a result of it, because (1) though the thrombosis of the vessels of the grey matter was most marked in the dorsal region at the

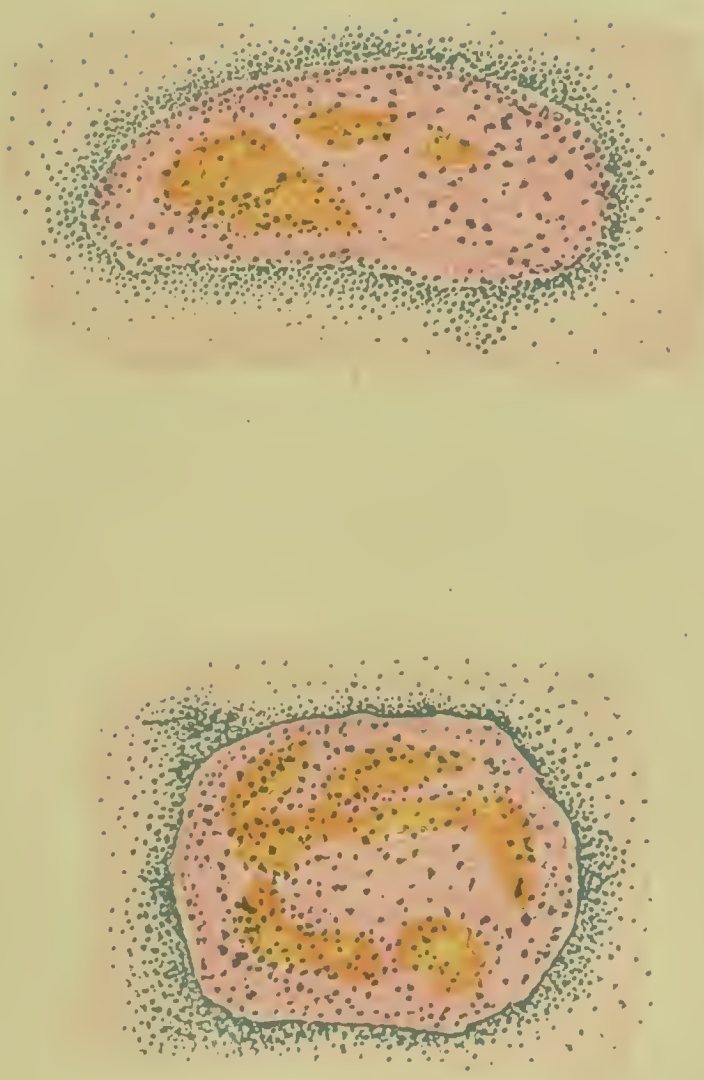

PLATE III.

Two dilated and thrombosed blood vessels in the grey matter of the spinal cord (Zeiss. D) Logwood and Eosin.

chief seat of hæmorrhage, and for a short distance above and below, yet thrombosed vessels were found in the meninges far away from the hæmorrhage, in the upper cervical and lumbar regions; (2) many of the thrombi were clearly of older date than the hæmorrhage; (3) there were marked vascular changes which alone would account for the thrombosis; and (4) the blood corpuscles in some of the thrombosed vessels were altered in shape, and some were broken down into granules, whilst the corpuscles in the hæmorrhage around were unaltered, and evidently the hæmorrhage was more recent than the thrombus.

The inflammatory changes—excess of nuclei and cell infiltration—were, as a rule, just around dilated vessels.

Though spinal thrombosis has been suggested by many writers as a primary cause of paraplegia of sudden onset in syphilitic patients, hitherto the pathological evidence has been slight. The pathological examination of the case recorded shows clearly that, in a syphilitic patient, paraplegia of sudden onset (with anæsthesia and bladder and rectal symptoms) was due to thrombosis in diseased vessels followed by hæmorrhage. The case is also of interest inasmuch as syphilitic disease of vessels and thrombosis are not usually included among the causes of hæmatomyelia in the discriptions of this disease.

Careful pathological records of cases of very acute syphilitic paraplegia are not common, and in order to form an opinion as to the pathological anatomy, I have arranged in tabular form (pages 64—66) the results of post-mortem examinations in a dozen of such cases recorded during the last ten years. These cases have not been specially selected, except that only those are tabulated in which the records are fairy complete, and in which the onset has been very acute.

A survey of the records tabulated shows that the disease has often run a very rapid course. In 7 out of the 12 cases it proved fatal within 32 days.

The seat of the chief changes in the cord has been the dorsal region; occasionally they have extended to the lumbar or cervical regions.

In 4 out of the 12 cases the meninges were only affected just around the vessels; here there was cell infiltration, but at other parts the meninges were normal. In

Author.	Journal.	Duration of Paralysis from onset to death.	Date of onset of Paralysis after syphilitic infection.	Region of Cord affected.	Meninges.	Meningeal Vessels.	Cord.	Vessels of Cord.	Other Changes.	Remarks.
Schmaus.	*Deutsches Arch. f. Klin. Med.* Bd. 44, 1889.	2 wks.	12-13 yrs.	Dorsal.	Only vascular changes (as in the Cord.)	Vascular changes same as in Cord.	Irregular patches of sclerosis in anterior and lateral columns around diseased arteries, best seen at periphery of Cord. Irregular patches of degeneration in the posterior columns.	Hyaline thickening of intima. Cell infiltration of intima. Periarterial cell infiltration. Thrombi in many of smaller vessels. Phlebitis also.		Degeneration of the Cord secondary to vascular disease.
Sottas.	*Paralysies Spinales Syphilitiques*, Paris, 1894	60 hrs.	2 yrs.	Chiefly upper Dorsal.	Normal, except cell infiltration around vessels.	Veins obliterated by thrombi or by concentiric narrowing of lumen. Phlebitis obliterans. All coats of veins affected. Changes in arteries less marked; in larger arteries external coat chiefly affected; in small arteries all the coats, but especially the external.	Softening and degeneration upper Dorsal region; other parts not affected.	Fibrous thickening and cell infiltration of walls of vessels; many obliterated. Veins more affected than arteries. Periarteritis more marked than endarteritis. Small gummata in walls of arteries. Perivascular infiltration. Thrombosis in some of vessels.	Few minute hæmorrhages.	Softening secondary to vascular disease, thickening and obliteration of vessels.
Sottas and Lancereaux	,,	5 mths.	7 yrs.	Dorsal & Cervical.	Meningitis.	Gummatous perivascular infiltration.	Necrosis in grey matter, also at periphery of Cord	Almost complete obliteration of spinal arteries; thrombosis: endarteritis.		Necrosis following arterial disease.
Sottas.	,,	15 mths.	14 mths.	Mid. and lower Dorsal.	Meningitis chiefly around vessels.	Endarteritis, periarteritis, phlebitis obliterans.	Softening and secondary sclerosis.	Endarteritis, periarteritis. hyaline degeneration, obliteration		Softening secondary to vascular disease.

Author.	Journal.	Duration of Paralysis from onset to death.	Date of onset of Paralysis after syphilitic infection.	Region of Cord affected.	Meninges.	Meningeal Vessels.	Cord.	Vessels of Cord.	Other Changes.	Remarks.
Goldflam.	*Wiener Klinik*, 1893. p. 65.	5-6 wks.	15 mths.	5th & 6th Dorsal; 12th Dorsal and 1st Lumbar.	Thickened; infiltrated with round cells, especially near vessels.	Walls thickened. Chiefly marked thickening of intima. Infiltration of adventitia; Vessels obliterated.	Degeneration of nerve elements; softening. Presence of compound granular cells.	Walls thickened; Chiefly marked thickening of intima. Adventitia infiltrated; Vessels often obliterated.	Blood extravasations around vessels in Dorsal part.	Softening following vascular changes.
——	p. 73.	32 days	1 mths.	Mid-Dorsal chiefly.	Thickening chiefly in Dorsal region. Cell infiltration chiefly around vessels.	Walls thickened. Obliteration common in veins. In arteries thickening of intima; Adventitia infiltrated.	Softening and degeneration of nerve elements.	Vessels full of blood. Walls thickened, but much less than those of pia-mater.	Small extravasations of blood.	Softening through vascular disease of pia-mater.
Rosin.	*Zeitschr. f. Klin. Med.* Bd. xxv., 1896	6 mths.	2 yrs.	4th & 9th Dorsal	Meninges not affected, except by cell infiltration just around vessels.	Arteries chiefly affected. Changes mostly in intima. Endarteritis, lumen narrowed. Vessels often thrombosed or obliterated.	Degeneration of nerve elements.	In some vessels endarteritis; lumen narrowed. Thrombosis in some vessels. In other vessels dilatation, walls thickened, especially adventitia, which is infiltrated with cells; intima little affected. Arteries chiefly diseased.	Small hæmorrhages.	Myelitis following vascular disease.
Möller.	*Archiv. f. Dermat. u. Syphilis*, 1891	2 mths.	18 mths.	Dorsal.	Normal, except cells just around vessels.	Endarteritis. Cell infiltration around vessels in some parts.	Degeneration and anæmic necrosis in white substance of lateral and posterior columns. Grey matter normal.	Intima of arteries chiefly affected. Endarteritis; other coats little affected. Endo-phlebitis, also thrombi in some vessels; hyaline degeneration of others.		Degeneration of Cord following arterial disease.

Author.	Journal.	Duration of Paralysis from onset to death.	Date of onset of Paralysis after syphilitic infection.	Region of Cord affected.	Meninges.	Meningeal Vessel.	Cord.	Vessels of Cord.	Other Changes.	Remarks.
Biernacki.	*Deutsche Zeitschr. f. Nervenheilk.* x., 1897. *Abstract Neurol. Centblt.*, 1897, p. 903.	23 days.	1 year.	3-5 Dorsal nerve root.	Not stated.	Not stated.	Disseminated patches of myelitis in lateral and posterior columns; old patches in posterior columns; secondary degeneration.	Endarteritis endo-phlebitis.		Softening following thrombosis and vascular disease; "myelopathia endo-arteriitica acuta."
Lamy.	*Nouvelle Iconographie De la Salpetriere*, 1893, p. 90.	19 days.	1 year.	Upper Dorsal.	Diffuse gummatous arachnitis and lepto meningitis in all regions.	Cell infiltration most marked about veins, gummatous phlebitis; obliterating endo-phlebitis; arteries almost unaffected	Softening in upper Dorsal region.	Thickening of vessel walls, lumen diminished, other vessels dilated. Distension of perivascular lymphatic sheath with round cells.		Softening associated with marked vascular changes.
Siemerling.	*Arch. f. Psychiatrie*, Bd. 22.	29 days.	(?)18 mths. to 2 yrs.	Lower Dorsal.	Pia-mater thickened with cell infiltration. Anterior roots surrounded by meningeal infiltration and atrophied.	Anterior spinal vein obliterated by thickening of the intima. Infiltration of adventitia of artery. Marked proliferation of intima of vertebrals. Infiltration of adventitia and muscular coat.	Gummatous mass of triangular shape extending from pia-mater into left anterior horn and anterior column in the lower Dorsal region. Patches of myelitic changes at lower Dorsal region.	Vessels thickened.		Marked vascular changes.
Williamson.	*Lancet*, July 7, 1894.	15 days.	2 years.	Mid. Dorsal.	Slight lepto meningitis, best marked in Dorsal region.	Endarteritis, narrowing of lumen, thrombosis, periarteritis; endo and peri-phlebitis; thrombosis.	Hæmorrhage; Degeneration; Grey matter chiefly affected.	Endarteritis, periarteritis, dilatation of small vessels, thrombosis.	Hæmorrhage extensive in grey matter.	Syphilitic disease of vessels. Thrombosis of spinal vessels. Secondary hæmorrhage & degeneration.

7 of the cases there was diffuse infiltration of the meninges with round cells—leptomeningitis. In 11 of the 12 cases, the vessels of the meninges showed marked changes—endarteritis, periarteritis, endo- and periphlebitis and phlebitis obliterans. The small arteries were often greatly narrowed by thickening of their walls, and sometimes thrombosis had occurred. Thrombosis was more frequently present in the veins. Occasionally small gummata were found in the walls of the veins or arteries. The changes in the meningeal vessels were sometimes best marked in the arteries, sometimes in the veins; sometimes the intima was more affected, sometimes the adventitia.

Both vascular changes and meningitis, when the latter was present, were best marked in the dorsal region, though they were seen at other regions also.

In the cord substances vascular changes, similar to those in the meninges, were present in all of the cases. Sometimes endarteritis or endophlebitis was the chief change; sometimes the adventitia was more affected. Often the vessels were obliterated, or almost obliterated, by the thickening of the walls. In other cases, the vessels were markedly dilated and full of blood, and the intima and muscular coat normal, whilst the adventitia was infiltrated with cells. In my own case thrombosis of dilated spinal vessels was a marked feature. In 5 of the other cases thrombosis was detected in some of the small spinal vessels (chiefly veins), but it was not such a prominent feature as in my own case. Hyaline and fibroid thickening of the small vessels was often present.

As a result of the vascular changes, there was softening or necrosis, and breaking down of the nerve elements. In some of the cases secondary ascending and descending sclerosis were present.

In my own case there was extensive hæmorrhage in the grey matter; in 4 of the other cases there was slight extravasations of blood.

In all of the cases the cord changes (softening, degeneration or hæmorrhage), were secondary to disease of the blood vessels.

The vascular disease was always of older date than the other changes. Infiltration with granulation or gummatous tissue was rare, and the changes were briefly those of softening or breaking down of nerve elements (so called myelitis), or hæmorrhage associated with syphilitic vascular disease in the cord or meninges, or both.

The vascular changes could not have developed suddenly, whilst the sudden paralysis can be fairly attributed to softening ("so called myelitis") or degeneration of nerve elements, due to deprivation of blood supply, or to these changes associated with hæmorrhage.

In all of the 12 cases the symptoms were evidently the result of pathological changes, caused by syphilitic disease of the vessels.

When the central arteries of the cord and their branches were most affected, the grey matter chiefly suffered; when the peripheral arteries, the white matter.

The meningitis, when present, took no real share in the production of the paralysis, but the disease of the meningeal vessels was probably the chief cause of the cord changes in a few of the cases.

Occasionally, on examination of cases of supposed acute syphilitic myelitis or paraplegia, the microscope has only revealed the appearances of ordinary idiopathic acute myelitis without any syphilitic vascular changes. I have not met with any case, well recorded, clinically and pathologically, in *recent* literature, in which these ordinary myelitic changes have been found, but such

cases have been reported, and I have made the pathological examination in one myself. But as Gowers suggests, it is possible that some of them have been instances of ordinary non-specific myelitis occurring in persons who happen to have suffered from syphilis previously.

Dejerine has described a form of acute central myelitis, which occurs at a comparately early date after the primary infection, and which runs a rapidly fatal course. (*Revue de Medicine*, 1884, p. 60).

A few cases of acute disseminated myelitis are on record, in which syphilis has been regarded as the cause.

VIII. ERB'S SYPHILITIC SPINAL PARALYSIS.

It has been pointed out by various observers years ago, that chronic syphilitic disease of the spinal cord may give rise to symptoms, more or less resembling those which are produced by an incomplete transverse myelitis in the dorsal region.

Charcot described such cases under the name of transverse syphilitic myelitis.

Ross and Rumpf have described similar cases.

Leyden stated in 1888, that weakness of the lower extremities with spastic symptoms, corresponding to a localised affection of the spinal cord, was the most frequent form of syphilitic spinal paralysis.

But it was not until 1892 that much attention was paid to these cases. In that year, Erb carefully described a class of cases of chronic spinal syphilis presenting a certain group of symptoms. He believes that these cases form a common and distinct clinical variety of syphilitic affection of the spinal cord.

The patient presents superficially the familiar symptoms of spastic paresis or paralysis, as regards gait, attitude, and movements. The patella tendon reflexes are increased, and ankle clonus is present, but there is relatively only slight muscular rigidity. The bladder is constantly affected. As a rule, there is only slight, though constant, affection of sensation. The onset of the disease is generally gradual, seldom rapid. Marked spastic paresis gradually develops, but only exceptionally is there complete paraplegia. The upper half of the body is unaffected. There is often a tendency to improvement under anti-syphilitic treatment.

During the last six years these cases have been the subject of much discussion, and the name of "Erb's syphilitic spinal paralysis" has been usually given to the affection. Many writers have recorded cases corresponding to Erb's description, and have confirmed his views as to the frequency of this clinical type of spinal syphilis. (Gerhardt, Marie, Sachs, Turner, Mitchell, Clarke, Kowalewsky, Trachtenberg, Nonne, and others). The whole subject has been exhaustively reviewed by Kuh, and Erb's cases recorded in detail. Trachtenberg has more recently made a complete compilation of cases on record, and has added others from the Cracow Hospital.

Oppenheim admits the frequency of the group of symptoms described by Erb. But he believes they are due to an ordinary syphilitic meningo-myelitis in the dorsal region, and that Erb's syphilitic paralysis is no disease *sui generis*. He thinks that the meningeal symptoms subside, and that partial myelitis and secondary degeneration remain. In the last edition of his text book on nervous diseases, Oppenheim describes Erb's group of cases, and states that the symptoms are mostly due to a syphilitic meningo-myelitis, localised chiefly in the dorsal region, and that they often correspond only to a definite stage of the affection.

Leyden and Goldscheider admit that Erb's group of symptoms is undoubtedly often met with after syphilitic infection, but that in absence of pathological records, there is not sufficient grounds for separating these cases from the other varieties of cerebro-spinal syphilis. They cannot admit that the group of symptoms are characteristic of syphilis, since they are met with in other forms of myelitis (localised, diffused and multiple). They believe that the relatively slight degree of muscular rigidity, with

great increase of the tendon reflexes, may occur also in disseminated myelitis. They think that Erb's group of symptoms is due to the localisation of the process to the dorsal region, and believe that they correspond only to a definite form and stage of development of spinal syphilis.

Whatever opinions may be held, as to the advisability of regarding Erb's group of symptoms as those of a special affection, there can be no doubt that cases of syphilitic disease of the spinal cord are not infrequently met with, which present this combination of symptoms. For clinical purposes it is convenient to class these cases together under the name of Erb's syphilitc spinal paralysis though the pathological basis is by no means clearly defined.

I have met with several cases, however, which have presented many of Erb's symptoms, but others have been absent, and a number of such cases have been recorded in literature.

Taking Erb's syphilitic spinal paralysis as a convenient clinical group, a sufficient number of cases have now been recorded to enable a detailed clinical description to be given.

(Trachtenberg collects most of the cases hitherto recorded in his able article in the *Z*eitschrift f. klin Med. Bd., 26, and from his description many details in this section have been taken.)

Ætiology.—The disease develops usually very soon after infection.

The date of onset in Erb's 22 cases was as follows:—

In the first 3 years	13 cases	
,, ,, 6 ,, ...	18	22 cases.
From 9 to 20 years	4	

Kuh's observations show that neither severe syphilis, nor careful treatment in the primary or secondary periods has any definite influence on the frequency of the occur-

rence of myelitis. He also states that the cord disease develops usually after the secondary symptoms.

In 56 cases analysed by Kuh (from literature and from Erb's clinic), 44 were between the ages of 20 and 41 years, and only 12 over the latter age.

Kowalewsky gives the usual age as 30 to 45.

As regards sex, in 62 cases analysed by Kuh, only six were females. Kowalewsky also states that it occurs chiefly in the male sex. The same author states that this type of spinal syphilis is common, but not so common as tabes dorsalis. In 152 cases of syphilitic disease of the nervous system, treated by him in 1892, 38 were tabes and 21 Erb's spinal paralysis.

Symptoms. — The onset of the affection is usually gradual: months, or even years elapse before the symptoms are fully developed.

Most frequently the patient complains first of a tired sensation in walking, and of weakness in the legs. Then bladder symptoms develop. Sometimes (as in the case recorded, page 76), the bladder symptoms occur first. Constipation is also an early symptom, and at the commencement, sometimes there is pain in the back.

It is stated that the symptoms may occasionally begin suddenly, but these cases are better excluded from consideration in this section.

When the affection is fully developed, motor symptoms predominate; there is weakness in the legs, and the movements are impared, but there is usually only partial paralysis—paraparesis, and complete paraplegia is rare. After a period of paresis, paraplegia occasionally developes suddenly, but if the case does not terminate fatally, the paraplegia subsides, and gives place to paresis.

The paraparesis is of the spastic form. The gait is spastic, but according to Kowalewsky this symptom is not

always present, and it is much less marked than in lateral sclerosis. The patient complains of stiffness of the legs. but there is only slight muscular rigidity as a rule.

The patella tendon reflexes are much increased; ankle-clonus is constantly present. The cremasteric, abdominal and plantar reflexes are sometimes diminished, sometimes lost. The motor symptoms and the increase of deep reflexes are often more marked on one side than the other, and sometimes one leg is affected only at first, and the other at a later date.

The arms are not affected.

Sensory disturbances are not usually marked, and do not extend over a large area.

Generally, there are only portions of the skin where the tactile and painful sensation is diminished. The temperature sensation is often altered or diminished markedly. Paræsthesia is also noted.

Kowalewsky has drawn attention to an increased temperature reflex in the legs, which he has observed in all of his cases; it is especially noticed on stimulation with warm objects; not unfrequently also by cold objects. Touching the legs with warm (or cold) objects, gives rise to very strong convulsive contractions. Kowalewsky states that he has not seen the symptom so marked in any other disease.

The bladder disturbances are the same when the disease is fully developed, as at the onset. Retention of urine or difficult micturition is present, and the use of a catheter is necessary. Sometimes a spastic incontinence of urine is observed. Weakening of the sexual power and constipation may occur.

Sometimes Rhomberg's symptoms is noted. Trophic disturbances do not occur in this stage, and the general nutrition is usually good.

As regards the course, improvement may occur, and finally, complete recovery may take place (according to Müchin, this occurs in 5 to 6 per cent.) Usually, however, recovery is not complete: the patient improves considerably, but still some weakness of the legs or bladder troubles remain. It is stated that partial recovery occurs in 20 to 25 per cent.

When the disease terminates fatally, the typical characters of the affection are lost: paraparesis passes into paraplegia; then bedsores and cystitis develop, and death occurs.

Diagnosis.—The spastic gait, the rigidity of the muscles, and the increase of the reflexes, are not so marked in Erb's paralysis as in lateral sclerosis (or, more correctly, cases in which there is sclerosis in the crossed pyramidal tracts). Also, bladder symptoms are present in Erb's paralysis, but not in lateral sclerosis. The difference is one of degree, as regards the spastic symptoms. The bladder affection is more diagnostic. Also sensory disturbances are present in the syphilitic affection. These points separate the syphilitic cases from amyotrophic lateral sclerosis also.

In transverse myelitis the paraplegia, the rigidity, and the sensory disturbances are greater, as a rule.

Briefly stated, the symptoms of Erb's spastic syphilitic paralysis appear to be those of a mild or partial transverse dorsal myelitis, as regards motion and especially as regards sensation, whilst the bladder symptoms are very prominent.

Of course, in all cases before making a diagnosis, the patient should be carefully examined for signs or symptoms of caries of the vertebræ.

Pathological Anatomy.—It is somewhat strange that in

this form of spinal syphilis, the published records of the pathological examination of the spinal cord are so few.

Erb stated in his early paper that probably the disease is due to a partially transverse lesion, *i.e.*, one not affecting the whole transverse section of the cord. He believed the lesion to be one involving the posterior halves of the lateral columns, the posterior grey matter and the posterior white columns.

Kuh regarded the changes as the result of syphilitic disease of the blood vessels.

Oppenheim, as already mentioned, thought that the change would be found to be a meningo-myelitis in the dorsal region.

The following is the record of a case in which I made a microscopical examination of the spinal cord.

During life the patient was under the care of Dr. Joseph, Honorary Surgeon to the Warrington Infirmary, and the abstract of the clinical history is taken from notes kindly furnished by Dr. Langdale, House-Surgeon to the hospital. Dr. Langdale also made the post-mortem examination, and to both of these gentlemen I am indebted for kindly forwarding the spinal cord to me for examination.*

Clinical History.—The patient was a man, aged 27, labourer, who first came under treatment in November, 1894. He had been a soldier, and had contracted syphilis in 1888, at the age of 21. In 1893 he began to be troubled with difficulty in micturition. This increased, complete retention occurred, and for almost twelve months before he first came under observation he had been obliged to use a catheter to draw off the urine.

When first examined, in 1894, he complained of retention of urine, of weakness of the legs, and of a tired feeling

* I have recorded this case in the *British Med. Journal*, December 31, 1898.

at the back of the knees on going upstairs. There was at that time no definite paralysis or wasting of the muscles of the legs. The plantar reflexes and the knee-jerks were increased: ankle clonus was present on the right side, doubtful on the left: the gluteal, cremasteric, and abdominal reflexes were present. There was no anæsthesia, no pain in the back, no girdle sensation and no inco-ordination. The pupils reacted normally, and the optic discs were normal.

Ten months later (in August, 1895) he began to suffer from pain in the back, especially at the lower part. In November, 1895, the legs were so weak that he was unable to walk without assistance. The movements of the left leg were greatly impaired, those of the right leg to a less extent. There was still complete retention of urine. The knee-jerks were increased, and ankle clonus was present on both sides. On the right leg, and on the abdomen on both sides as high as the umbilicus there was slight impairment of cutaneous sensibility: on the left leg sensation was normal. The urine contained pus. After rest in bed the condition of the bladder improved, and he was able to pass urine without the aid of a catheter. The legs also improved rapidly, and the patient was able to walk again, but the gait was unsteady. Sensation became quite normal.

In December, 1896, the patient again came under observation. There was then some difficulty in micturition. He was able to walk, but there was inco-ordination of the lower limbs. At a later date there was obstinate vomiting and hiccough, and the urine contained pus. Death occurred in February, 1898.

The post-mortem examination was made by Dr. Langdale, who kindly sent me the spinal cord and portions of the brain for examination. The necropsy revealed

extensive double pyelonephritis, but no other macroscopic changes of interest were detected. The cerebrum, cerebellum, pons and medulla appeared normal macroscopically.

Pathological Examination.—The spinal cord was hardened in Müller fluid; numerous pieces were embedded in celloidin. Sections were prepared and stained according to Weigert's method, and with logwood and eosin, and with aniline blue black. Diagram 1 represents the distribution of the changes in the spinal cord at various levels. The portions of the figures in this diagram which are shaded by dots, represent the regions of degeneration and secondary ascending and descending sclerosis—that is, the parts in which, in the sections stained according to Weigert's method, nerve fibres are absent or scanty.

Microscopical Examination.—In the pia mater and arachnoid evidences of slight meningitis were found in all regions of the cord, but the changes were most marked in the dorsal region, least marked in the cervical region. The meninges were slightly thickened by fibrous tissue, and in various parts there was slight infiltration, with round cells chiefly around the blood vessels.

The spinal blood vessels presented well-marked changes in numerous parts. The larger arteries in the meninges, especially at the posterior part of the cord, near the posterior nerve roots, often presented marked endarteritis. The internal coat was often much thickened by abundant new cell formation on the inner side of the elastic lamina, the lumen of the vessel being thereby much diminished (see Diagram 2). The middle and external coats were generally not much altered; but frequently there was an infiltration of leucocytes around the vessels. The veins were either unaffected or the changes (proliferation of the intima cells) were less than in the arteries. In many

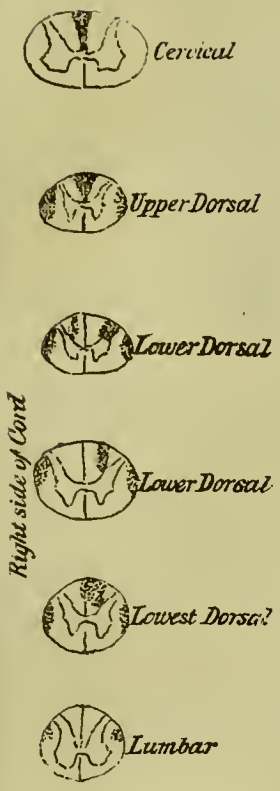

Diagram 1.

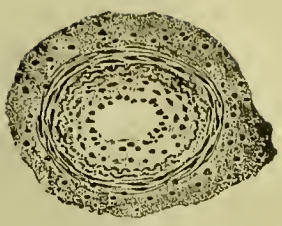

Diagram 2.

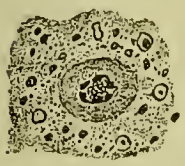

Diagram 3.

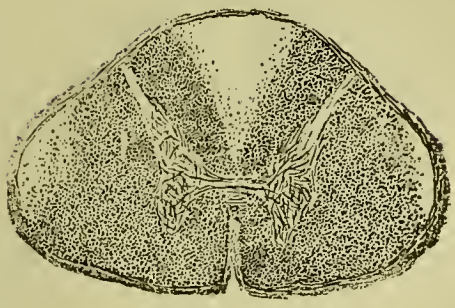

Diagram 4.

Diagram 1.—Showing distribution of lesions in the spinal cord at various levels. The sclerotic and degenerated parts are shaded. Diagram 2.—Section of meningeal artery showing endarteritis. Diagram 3.—Section of small vessels in the white matter of the cord showing hyaline thickening of its walls. Diagram 4.—Section of spinal cord in upper dorsal region. Weigert's stain. Pale parts = regions of sclerosis. Black dots = normal nerve fibres.

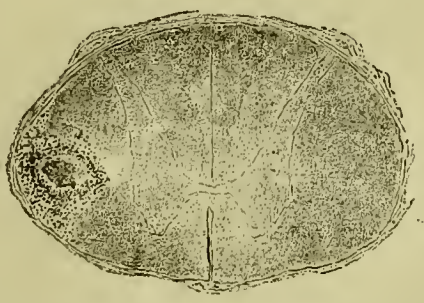

Diagram 5.—Representing section stained with logwood and eosin. Gummatous mass surrounded by cell infiltration, extending inwards from the surface of the cord. Upper dorsal region.

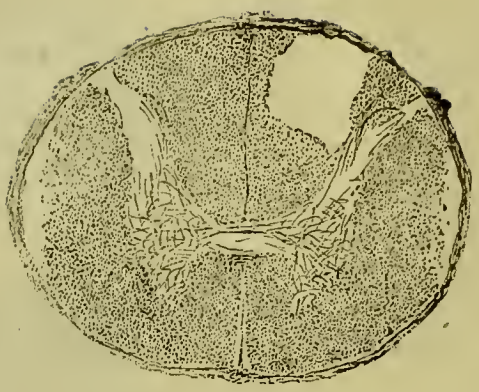

Diagram 6.—Section of spinal cord in lowest dorsal region (Weigert's stain); affected parts pale; normal nerve fibres represented by black dots.

places the veins were surrounded by cell infiltration. The very small meningeal arteries presented thickening of the walls and slight proliferation of the cells of the intima, and they were often surrounded by round-celled infiltration.

The meningeal vessels were generally well filled with red corpuscles, but in a few places the lumen of a vessel was partially occluded by a thrombus.

Within the cord, especially in the diseased parts indicated on p. 79, the walls of the small vessels were often very much thickened,, and had a homogeneous hyaline appearance, also the endothelial cells of the intima were slightly proliferated (see Diagram 3). In some places these hyaline vessels were surrounded by slight infiltration of leucocytes.

The two prominent features of the vascular changes were the endarteritis of the larger meningeal arteries and the hyaline thickening of the walls of the small vessels within the cord.

The changes in the white matter will be best indicated by describing the sections at various levels.

In the cervical region there was marked degeneration in the posterior median columns, and the degenerated

region presented the usual appearances of secondary sclerosis, namely, absence of nerve fibres and increase of neuroglia connective tissue. The pia mater was slightly thickened by fibrous tissue, and in the lateral region of the cord, just beneath the pia mater, in the region of the direct cerebellar tract, and a short distance in front of it, there was an increase in the neuroglia connective tissue and a diminution of the number of nerve fibres. But this peripheral sclerotic change was very slight, and did not extend to the crossed pyramidal tract. The last mentioned tract and all other parts of the white matter appeared normal. The grey matter was normal.

In the upper dorsal region marked degeneration was seen in the posterior columns, localised to the posterior median column and a little of the adjacent part of each posterior external column. The sclerosis at the anterior part of this region—that is, close to the grey commissure was slight. In the lateral region of the cord there was a peripheral strip of degeneration just under the thickened pia mater. The degeneration extended inwards, and affected the outer part of each crossed pyramidal tract (see second figure, Diagram 1, and also Diagram 4). The degenerated parts, both in the posterior and lateral regions, presented the ordinary appearances of secondary sclerosis—namely, disappearance or diminution of the nerve fibres and great excess of neuroglia connective tissue with dilated blood vessels.

The sclerotic changes were more marked in the lateral column of the right side, and at one point on this side there was a gummatous infiltration. This commenced from the pia mater and surface of the cord, and extended inwards as an irregular, somewhat oval mass, just anterior to the position of the crossed pyramidal tract (see Diagram 5). In the centre of the patch was a small mass of

amorphous caseous substance. This was surrounded by nucleated fibrous tissue, and externally was a zone of abundant cell infiltration. The small vessels presented marked hyaline thickening of their walls. In some of the sections this gummatous infiltration was seen to invade the grey matter slightly.

In the mid-dorsal region there was a similar distribution of the peripheral sclerosis in the lateral columns (as in Diagram 4). But in the posterior colums the intense sclerosis in the region of the posterior median columns had disappeared. There was, however, a slight diffuse increase of the neuroglia fibrous tissue all over the posterior columns, both external and median. The neuroglia was well supplied with nuclei, and the small blood vessels were dilated. Nerve fibres were seen at all parts of the posterior columns in this region, but their number was much less than in a healthy spinal cord, and they were more widely separated by neuroglia connective tissue. The grey matter was normal.

In the lower dorsal region there was a small degenerated sclerotic patch in the posterior columns adjacent to each posterior horn of grey matter (as shown in the third figure of Diagram 1). The one on the left side implicated the grey matter of the posterior horn. There was also a peripheral zone of sclerosis in each lateral region. At a lower level only one of the sclerotic patches in the posterior column was seen (as shown in the fourth figure of Diagram 1). In the lowest dorsal region, just above the lumbar enlargement, there was a large patch of degeneration in the posterior columns of the left side. It extended from the posterior median fissure up to the posterior horn of grey matter (as shown in the fifth figure of Diagram 1 and also in diagram 6). In this degenerated patch, sections stained according to Weigert's method

showed a complete absence of nerve fibres. The periphery of the patch was bounded by a broad zone of dense fibrous tissue. Internal to this zone was a region in which there was a considerable number of blood vessels with hyaline walls, surrounded by a network of well-nucleated fibrous tissue. In the centre of the patch were numerous tortuous blood vessels, presenting marked hyaline degeneration of the walls, and surrounded by round-celled infiltration. This cell infiltration varied in amount at different parts; it consisted of leucocytes, together with a few compound granular cells, but none of the large epitheloid-like cells, so common in acute non-syphilitic myelitis, could be detected. In the lateral part of the cord at this level there was the same peripheral zone of degeneration (Diagram 6) as in the mid-dorsal region.

In the lumbar region of the cord the only change, apart from the affection of the meninges and their vessels (described above), was sclerosis, just limited to the region of the crossed pyramidal tracts (as shown in the last figure of Diagram 1).

Grey Matter of the Cord.—As mentioned above, at one point in the lower dorsal region a sclerotic patch invaded the whole of the posterior horn of grey matter; also at one spot in the upper dorsal region the outer half of the grey matter was invaded by gummatous infiltration on the right side; but in all other parts of the cord the grey matter was normal, with the exception of dilated and diseased blood vessels. The nerve cells and fine nerve fibres of the grey matter seen in Weigert's specimens were everywhere normal, except at the points just mentioned. The columns of Clarke appeared normal.

As regard the distribution of the changes above described, it is evident that the degeneration in the posterior median

columns in the cervical region was of the nature of secondary ascending sclerosis; it was less extensive than that in the posterior columns of the upper dorsal region. The degenerative changes in the lateral region of the cord were evidently due to a sclerosis produced by the extension inwards of inflammatory changes beginning in the pia mater. As mentioned above, they affected the peripheral region of the cord, and also implicated the crossed pyramidal tract. In the lumbar region, however, there was distinct secondary descending sclerosis, exactly limited to the crossed pyramidal tract.

A prominent feature of the pathological changes was the endarteritis of the larger vessels, especially in the meninges and the hyaline degeneration of the smaller vessels. All the degenerated portions of the cord presented sclerotic changes except those in the posterior columns in the lowest dorsal region, and at one point in the antero-lateral columns on the right side in the upper dorsal region. Here the changes were those of a gummatous infiltration.

The pathological changes were certainly not those of disseminated sclerosis; there was also marked secondary ascending sclerosis in the cervical region, which is very rare in disseminated sclerosis; and during life the symptoms were not those of the disease just mentioned. The typical endarteritis, the slight meningeal changes, the extension of the lesion from the meninges into the adjacent parts of the cord, the histological (gummatous) nature of the most recent patches, and the syphilitic history of the patient, all indicate clearly that the case was one spinal syphilis.

The pathological and microscopical examinations of the spinal cord, which have hitherto been recorded in cases of Erb's syphilitic spinal paralysis, are too few to

enable one to draw definite conclusions as to the lesions, and possibly they may not always be of the same nature and distribution. But the above record furnishes a good example of the changes which may be met with, in a case which presented during life most of the symptoms of "Erb's syphilitic spinal paralysis." The chief point of difference from Erb's cases was the gait; there was considerable inco-ordination in the movements of the legs; but this has been recorded in other cases also.

Briefly the pathological changes may be summarised as follows: Endarteritis and hyaline degeneration of the arteries of the spinal cord and meninges; slight meningitis: gummatous infiltration of the right antero-lateral columns in the upper dorsal region; sclerosis of the periphery of the cord in the lateral columns in the whole of the dorsal region; sclerosis in the posterior median columns in the upper dorsal region; irregular sclerotic patches, with one patch of cell infiltration (gummatous) in the lowest dorsal region; descending sclerosis in the lumbar crossed pyramidal tracts; ascending sclerosis in the cervical posterior median columns.

Kuh has recorded a case in which a post-mortem examination was made, but unfortunately I have not been able to see the original article and could only obtain information of the macroscopic changes. There was disease in the cord, most marked between the 8th and 10th dorsal segment. The lateral columns and Goll's columns were chiefly affected.

A few other cases are on record in which pathological examinations have been made, but as nearly all of them have been criticised I have thought it better to omit further reference to them.

Cases presenting degeneration in the lateral and posterior columns.—A few cases of chronic syphilitic paraplegia have been published, some before and some after Erb's type of spinal syphilis was recognised, in which a pathological examination has been made. During life the symptoms have usually *not* quite corresponded to Erb's description, and on examination the cord has presented changes in the posterior and lateral columns.

The following are the notes of a case which I published in 1891. The symptoms indicated a lesion of the dorsal part of the cord, but they differed from Erb's description in several points. The case was reported simply as one of syphilitic paraplegia, and not as Erb's paralysis; and the changes were chiefly in the lateral and posterior columns.

Changes in the Spinal Cord in a case of Syphilitic Paraplegia: Sclerosis of the Lateral Pyramidal Tracts and Goll's Columns, with Peripheral Sclerosis.

The patient had suffered from complete paraplegia, with rigidity of the limbs and bladder symptoms, for nine years.

Unfortunately I have no notes of the case taken during life, but Dr. H. Wilson, of Cheadle, to whom I am indebted for the opportunity of examining the cord, attended the patient on several occasions, and has kindly given me the following very important details with respect to his symptoms:—

A. B. was formerly a strong well-built man. He contracted syphilis in 1880. He was treated by a medical man, and the chancre healed in about three weeks. About one month later he noticed "some round yellowish-red spots on his body." Several months after he contracted syphilis, retention of urine occurred somewhat suddenly, and was followed by loss of power in the legs. The patient became almost completely paralysed. He was admitted as an in-patient at the Manchester Royal Infirmary, but, after a few days, was sent to the Cheadle Convalescent Home. In a short time the paralysis rapidly disappeared; the patient was able to walk again, and left the hospital. But a few weeks later the weakness returned rather suddenly, and the legs became completely paralysed. They remained in that condition until his death in 1890, a period of about nine years. He began to suffer from incontinence of urine and fæces at the onset of the second attack of paraplegia, and the incontinence continued up to the time of his death. In the course

of time the legs became rigid. He came under Dr. Wilson's care in 1885, and at that time there was well marked rigidity of the legs. The right leg was rigidly flexed on the thigh, and the thigh on the abdomen. The left leg was often flexed in a similar manner, but was sometimes extended. He could pull up the left knee with his hands, when the leg was extended, but had great difficulty in getting the limb extended again. When the left leg was extended, stroking the skin of the scrotum would cause it to become flexed. He did not suffer from pain in his legs, nor did he complain of numbness. During the time Dr. Wilson attended him, he could always feel quite well when his legs were pinched. There was no spinal curvature. Patient complained at times of slight pain in the lumbar region of spine.

For a long time before his death his urine was ammoniacal, and contained a large quantity of pus. He passed a catheter for himself once a week. For several years before his death he suffered from constipation, his bowels being moved only once a week.

The patient was a sharp, intelligent man, and supported himself by framing pictures and retailing pots, pans, etc. He was frequently to be seen driving about the country, in a low cart. He made his last excursion (into the Potteries) in June, 1890, and caught a severe cold through lying in his wet clothes.

He was seen by Dr. Wilson on July 5th, and then complained of great pain at the epigastrium, nausea, and vomiting, and frequent eructations. The face was anxious and pinched. Pulse between 80 and 100. Tongue furred. Bowels constipated, and urine ammoniacal (as had been the case for a long time). The pupils were normal and reacted to light. Temperature was normal. There were no girdle pains. The legs were flexed, as mentioned above. There was a large bed sore over the sacrum, which had existed for some time. The intellect was perfectly clear up to a few minutes before death. All the remedies resorted to failed to relieve the epigastric pain. The patient died on July 12th (1890). His attendant was raising him in bed when death occurred suddenly.

The patient had never suffered from hemiplegia, and there were never any cerebral symptoms. The patient's arms were never paralysed nor affected in the least. He was very skilful with his hands in framing pictures, etc. He was able to write quite well, and his speech was never affected.

The post-mortem examination was made by Dr. Reynolds, at the patient's house.

The bladder was much distended, and contained purulent urine. The ureters were dilated enormously, and the walls very thick. There were numerous abscesses in both kidneys.

After removing the spinal cord, a number of transverse sections were made, but no patch of transverse myelitis could be detected by the naked eye, and

the cord was preserved for microscopical examination. On examination of the membranes and the surface of the cord, nothing abnormal was detected. The cord was hardened in Müller's fluid, pieces embedded in celloidin, and sections cut and stained with aniline blue-black, with Weigert's hæmatoxylin, and with logwood.

Microscopical Examination—Upper Cervical Region.—Sections of this region, stained according to Weigert's method, showed well marked degeneration of the whole of both columns of Goll (from the posterior grey commissure up to the surface of the cord). There was marked degeneration of both lateral pyramidal tracts and of the direct cerebellar tract. The degeneration also passed forwards along the periphery of the cord, occupying the regions of antero-lateral ascending tracts of Gowers. A line drawn transversely across the cord, so as to pass through the most anterior part of each anterior grey horn, would give the limit of the extension forwards of this peripheral degeneration. (See plate IV.) In the above-mentioned tracts the degeneration was exceedingly well-marked, the tracts in the Weigert's specimens being of a deep brownish yellow colour, and each containing only a very few scattered remnants of nerve fibres (stained black) mostly at their periphery.

In sections stained with aniline blue black, the degenerated tracts just mentioned were deeply stained.

Under a high power the sclerosed tracts showed an enormous increase of the neuroglia fibrous connective tissue, in which oval or circular spaces were seen—spaces from which the nerve fibres had entirely disappeared. Around the margins in each sclerosed tract, a few scattered nerve fibres were seen, and at other parts, only the remaining axis cylinders of the nerve fibres; but in the centre of each sclerosed tract the nerve fibres had completely disappeared, and nothing remained but neuroglia connective tissue with oval or circular spaces. In a few of these spaces round cells were seen.

In the sections stained with logwood, the neuroglia fibrous tissue of the sclerosed area was seen to be well supplied with nuclei.

The blood vessels of the cord were dilated, especially in the sclerosed tracts (lateral pyramidal and Goll's), also to a less extent in the grey matter. In the crossed pyramidal tract, in the cervical region especially, they were exceedingly dilated. The walls of the vessels were much thickened, especially in the degenerated parts, and most markedly in the crossed pyramidal tracts. There was slight proliferation of the endothelium cells of the intima, but the increase in the thickness of the vessel walls was due almost entirely to marked fibrous thickening of the external coat.

In the *middle cervical region* the tracts of degeneration were much the same (see plate IV.)

In the *lower cervical region* only the anterior two-fifths of the left columns

PLATE IV.

SYPHILITIC PARAPLEGIA. WEIGERT'S STAINS.

Sclerosis in posterior and lateral columns (sclerosed parts pale brown). Figures arranged in the following order :—

Upper cervical region.	Dorsal region.
Middle cervical region.	Dorsal region.
Lower cervical region.	Dorsal region.
Junction of cervical & dorsal.	Lumbar region.

of Goll were degenerated and the anterior one-third of the right column of Goll. The other tracts of degeneration were the same as in the upper cervical region. Burdach's columns were not affected. In sections made a little lower, the degeneration in the columns of Goll did not extend so far towards the posterior surface of the cord. It extended anteriorly, however, almost up to the posterior commissure. The degeneration of nerve fibres was also much less complete than in Goll's columns at a higher level.

The degeneration of Goll's columns diminished gradually from above downwards, until about the junction of the cervical and dorsal regions only a small area of degeneration could be found in the left column of Goll at its anterior parts (see plate IV.) Burdach's columns were unaffected.

In the *dorsal region* there was marked degeneration of the lateral pyramidal tract. In the upper dorsal region a patch of degeneration ran also along the periphery of the cord from the posterior cornua; its anterior limit would be bounded by a line drawn across the cord through the anterior white commissure. Burdach's and Goll's columns were unaffected (see plate IV.)

In the *lumbar* region only the lateral pyramidal tracts were degenerated. (See plate IV).

The nerve fibres and cells of the grey matter were present in all the regions of the cord. The nerve cells of the anterior horns were quite normal in appearance in the lumbar and dorsal regions, but they were somewhat shrunk in the cervical region.

In a few of the sections from the middle cervical region there were occasionally a few small irregular patches of sclerosis in Burdach's columns, just adjacent to the complete degeneration of Goll's columns, but Burdach's columns were almost unaffected throughout the whole length of the cord. The whole of the cord was cut into section, from the upper dorsal region (fifth fig. in plate IV), where there was no affection of either Goll's columns or Burdach's columns, up to the middle cervical region, where both columns of Goll were completely degenerated. In all of these sections Burdach's columns were unaffected.

In a few of the sections from the cervical region the sclerosis just at the periphery of the cord was prolonged forwards up to the anterior median fissure, and there was also a small tract of sclerosis just in the centre of each direct pyramidal tract (Türck). Both above and below the level of these sections the direct pyramidal tracts were not affected.

No complete area of transverse myelitis was discovered in any part of the cord.

The pia-mater was normal in the lumbar and dorsal regions. In the cervical region it was thickened, the increase in thickness being due to an

increase of fibrous tissue. The blood vessels of the pia-mater were slightly dilated.

The changes in the spinal cord, in the case just recorded, differ from the ascending and descending degenerations following a transverse myelitis, in the following points :—

(1) Just above the lesion in a transverse myelitis, or other transverse lesion, the degeneration is not confined to Goll's columns, but exists also in the posterior external (Burdach's) columns; the more extensively the nearer to the lesion, until immediately above the lesion, the whole of the posterior columns (median and external) are degenerated, except a very small area close to the posterior cornua.

In the case just recorded, the posterior external column was almost entirely free from degeneration throughout the whole length of the cord. At no part were the two columns both extensively degenerated, as occurs just above the lesion in the case of a transverse myelitis.

(2) In the case recorded, on making sections of the cord from below upwards, the degeneration in the posterior columns was first found in the anterior part of Goll's columns, about the junction of the dorsal and cervical regions, and at this level there was no degeneration in the posterior external columns. This degeneration gradually increased in extent from below upwards, until both of Goll's columns were completely degenerated. These columns then remained degenerated up to the highest part of the cervical region, whilst the posterior external columns remained practically unaffected.

In ascending degeneration following a transverse myelitis, the degeneration in Goll's columns is most marked just above the lesion, and gradually diminished toward the upper part of the cord. If the lesion be in the dorsal or lumbar region, then the degeneration in Goll's columns is limited to the posterior two-thirds of the columns, above the middle of the cervical enlargement.

(3) In transverse myelitis the crossed pyramidal tracts are degenerated below the lesion and Goll's columns above the lesion. Thus, at the part where the crossed pyramidal tracts are degenerated, Goll's columns are not degenerated, and *vice versâ*.

In the case above described the crossed pyramidal tracts are degenerated throughout the whole length of the cord. Hence, in the cervical region both posterior median and crossed pyramidal tracts are degenerated.

(4) No area of transverse myelitis could be discovered, though the whole of the cord was cut into sections from the upper dorsal region (where both posterior columns were unaffected) to the middle cervical region, where Goll's column was completely degenerated.

Hence the changes in the cord are quite different from those which occur secondarily to a transverse myelitis.

There is nothing in the history pointing to a cerebral lesion. The patient had no cerebral symptoms throughout the entire course of the illness.

In the case recorded above, the following are the chief points of interest: (1) The pathological changes in the cord :—(*a*) Sclerosis of the lateral pyramidal tracts throughout the whole length of the cord. (*b*) Sclerosis in Goll's columns from about the junction of dorsal and cervical regions up to the highest part of the cervical region. [The sclerosed area in Goll's columns is small at first, gradually increasing for a little distance upwards, and then the whole of the column is degenerated up to the highest cervical region.] (*c*) The sclerosis in the direct cerebellar tract, beginning in the upper dorsal region and extending to the highest cervical region. (*d*) Sclerosis at the periphery of the cord in the cervical region, extending further forward than the direct cerebellar tract. (*e*) The absence of any, except the slightest changes, in the posterior external column. (*f*) The absence of any area of transverse myelitis.

(2) The distinct history of syphilis, and the great probability (almost certainty) that the paralytic symptoms were due to syphilis.

(3) The sudden onset of paraplegia, accompanied by bladder symptoms from the first; afterwards, spastic condition of the limbs—spastic spinal paraplegia.

It appears very probable that the sclerosis in the posterior and lateral columns was really started by a meningo-myelitis.

Other cases are on record in which changes have also been found in the posterior and lateral columns.

Nonne reports two :—

In his first case the patient was a man aged 50, who had suffered from syphilis at the age of 32. Spastic paresis developed gradually in the legs, the tendon reflexes were increased, the muscular rigidity was slight. Sensation was not affected. The bladder was only slightly affected; urine was passed slowly. Death occurred from cancer of the lip. The pathological examination of the spinal cord revealed a combined system disease. There was degeneration of the direct cerebellar tract and of Goll's columns. At the upper part of the cord the crossed pyramidal tracts were also degenerated; but mostly so at the lower part of the cord. There were no changes in the vessels, meninges or ganglion cells.

In Nonne's second case the patient was a man aged 45, who had suffered from syphilis. Ten years after the infection, spastic paresis developed gradually. Then bladder symptoms appeared, and afterwards cystitis. There was slight affection of sensation; the muscular rigidity was for a long period very slight, and only became marked towards the end of the

disease. The upper extremities were unaffected. The microscopical examination revealed a chronic transverse myelitis, which had affected the lateral columns, the posterior columns, and the posterior horns slightly; whilst the anterior horns and anterior white matter was unaffected. Also the crossed pyramidal tracts and the direct cerebellar tracts were degenerated *above* the lesion up to the upper cervical region, and the same regions of the cord were degenerated *below* the lesion. Above the dorsal region there was ascending degeneration in Goll's column.

Nonne states that in his cases there was a combination of a chronic dorsal myelitis, with ascending and descending degeneration, and a primary disease of the lateral pyramidal and the direct cerebellar tracts. There was no indication of meningitis, vascular disease, or syphilitic infiltration of the cord.

In both of Nonne's cases there was a very long interval between the syphilis and the spinal symptoms; and the cases during life did not exactly correspond with Erb's description.

Eberle (quoted by Nonne) records the case of a man aged 50 who had a hard chancre 15—20 years before the onset of symptoms. He complained of weakness of the legs, with stiffness; spastic paresis developed, with increase of the reflexes, but the muscular rigidity was not marked. There was slight diminution of sensation, and paresis of the bladder. The microscopical examination revealed degeneration of the crossed pyramidal tracts from the lumbar region to the pons. Also the posterior columns (Goll's and Burdach's) were degenerated.

Nonne draws attention to another case. Westphal records the case of a patient whose symptoms developed three years after a syphilitic infection. There was a gradual onset of spastic paresis of the legs, with increase of the reflexes. The muscular rigidity to passive movement was slight for four years. The sensory disturbances were also slight. The bladder at first was not much affected, but later marked bladder symptoms were present. Examination of the cord revealed degeneration of the crossed pyramidal tracts, the direct cerebellar tracts, and the periphery of the white matter of the cord. There was also primary degeneration of Goll's column in the dorsal and cervical regions.

In the case which I have recorded (p. 86) there can be little doubt that the changes were produced by syphilis, and in the other cases just mentioned, syphilis was probably the cause of the cord lesion.

Clinically in all of the cases, the symptoms were those of spastic paraplegia, indicating a lesion in the dorsal

region, and occurring in individuals who had suffered from syphilis. In all of the cases there was degeneration in the posterior and lateral columns. In the posterior columns the degeneration was chiefly in Goll's column at the upper part of the cord. In the lateral columns the crossed pyramidal tracts were degenerated in all regions of the cord; the direct cerebellar tracts in the cervical and upper dorsal regions.

Nonne and Trachtenberg believe that the degeneration of these columns of white matter is caused by a syphilitic toxine.

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IX. GUMMATA OF THE SPINAL CORD AND MENINGES.

Distinct macroscopic gummata, in the form of circumscribed tumours of the meninges, or of the cord, are very rare; but a number of cases are on record in medical literature. The diagnosis is usually based on the clinical history, and pathologically the condition is very seldom met with. Frequently other syphilitic changes are present in the spinal cord, and the symptoms produced thereby are combined with those due to the gumma.

When the gumma has commenced in the meninges, the symptoms have been, in many cases, the same as those of a non-specific meningeal tumour. There have been pain and symptoms of irritation of nerve roots first, and paraplegia at a later date. When the gumma has commenced within the spinal cord, the root symptoms have been usually absent, and clinically the case has presented the symptoms of a rather slowly developing paraplegia. A diagnosis of gumma has been frequently made when a syphilitic patient has developed the symptoms of a unilateral lesion—Brown-Séquard's paralysis; but such cases frequently improve under anti-syphilitic treatment, and the diagnosis has rarely been confirmed pathologically.

Gummata in the meninges are more frequent than those in the cord, and in the latter affection the gumma has frequently developed from the pia mater first, and then invaded the cord, though this is not always the case. Occasionally disseminated miliary gummata of the meninges have been found post-mortem.

The following is an abstract of notes of the pathological condition, and of the clinical symptoms, in a case in which I found a gumma of the spinal cord :—

Jane M., aged 41, married, was admitted as an in-patient, on account of paraplegia, at the Manchester Royal Infirmary, on March 20, under the care of Dr. Graham Steell, to whose kindness I am indebted for the opportunity of making the pathological examination of the cord, and of reporting the case.

Previous History.—About three months previous to admission, whilst getting through a half-opened window, the patient "twisted" her back and struck it against the bottom of the window sash. The part hurt was the dorsal region, and the pain was so severe that she fainted at the time ; but she soon recovered, and felt quite well for a few days. Then she began to suffer from pain in the back, which continued more or less until admission. At times the pain has been *very severe*. It has been situated *chiefly* "between the shoulders." Sometimes there has been pain in the lumbar region also. A girdle sensation was noticed about two weeks before admission, and also a sensation of "numbness" in the legs. Loss of power in the legs developed on March 16. Two days later bladder symptoms first appeared. A definite history of syphilis was not obtained, but it was exceedingly probable that the patient had been exposed to the risks of syphilis.

On admission the patient was unable to perform any movement of the legs. There was no localised wasting of the legs, and no rigidity. The knee jerks and plantar reflexes were present, but there was no ankle-clonus. There was no pain in the legs, and no tenderness of the muscles. There was *complete* anæsthesia of the right leg, but *only partial* anæsthesia of the left. The firm touch of a pin's head was not felt on the *right* leg ; also a deep pin prick was uot felt on the right leg. On the *left* leg a fairly firm touch with a pin's head was felt ; also a pin prick, if fairly deep, was felt ; but neither was localised correctly. The patient could distinguish between the head and point of a pin on the *left* leg, if fairly firm contact were made. The head and point of a pin were not felt on the lower half of the abdomen, but both were felt about 1½ inch above the umbilicus. The abdominal and epigastric reflexes were absent. There was retention of urine, with dribbling, and it was necessary to use the catheter twice daily. There was also loss of control over the motions. The arms and cranial nerves were not affected. There was tenderness on percussion of the spine from the fourth to the seventh dorsal vertebra, but there was no spinal curvature. Rhonchi were heard over the greater part of both sides of the chest. Heart, normal.

March 25. Tenderness in the region of the sixth dorsal vertebral spine.

April 4.. Urine contained a large quantity of pus. Slight plantar reflexes

were obtained on both sides. The knee jerk was present on the right side; absent on the left. There was no ankle clonus.

April 15. Right knee jerk exceedingly feeble; left, absent. No ankle clonus. Plantar reflexes present. No rigidity of the legs. Marked anæsthesia to tactile and painful impressions on *both* legs. The breathing gradually became more difficult. The pulse became more rapid, and numerous small râles were heard on both sides of the chest.

April 17. Pulse, 132; respiration, 34; temperature, 101°. Death occured on April 20.

Treatment.—Antisyphilitic from the first; spirits of ammonia and senega; and afterwards chlorodyne, etc., for the bronchitic symptoms and complications.

Autopsy.—Heart somewhat soft and flabby. Pleuritic adhesions and bronchitis on both sides. Pneumonic consolidation of the lowest lobe of the right lung. Abdominal organs presented no changes of importance. The spinal cord was distinctly soft and gelatinous in appearance about the level of the eighth dorsal vertebra. The membranes were normal to the naked eye.

The spinal cord was hardened in Müller's fluid. Naked eye examination showed, on section, a pale diseased area in the lower dorsal region, chiefly on the left side. This region was cut into small blocks, which were embedded in celloidin. Sections were stained according to Weigert's method, also with logwood and aniline blue-black. On the left side of the cord was an area of myelitis, having a longitudinal extent of nearly one inch. It involved the white matter of the cord in the lateral region and also the grey matter on the left side. At one point, about the middle of the longitudinal extent of the myelitis, was a small tumour about the size of a small pea, which involved the left posterior horn of grey matter and the adjacent white matter of the lateral column. The tumour could be seen distinctly with the naked eye on section of the cord. Microscopically, it consisted of amorphous caseous material, and was surrounded by a broad capsule of fibrous tissue; also a tract of fibrous tissue ran across the caseous material, as shown in the diagram. No giant cells, or other cell structure, could be seen within the caseous portion of the tumour. The zone of myelitis around the fibrous capsule was studded with numerous greatly dilated blood vessels, and infiltrated with compound granular cells and leucocytes. At one point the myelitis extended across the commissure and invaded the grey matter of the right half of the cord; also at one point the myelitis on the left side extended forwards in the white matter, just in front of the grey anterior horn, up to the anterior surface of the cord.

When sections of the tumour were treated with dilute hydrochloric acid and ferrocyanide of potassium, no signs of iron-containing granules could be detected. The pathological diagnosis rested between a small syphilitic

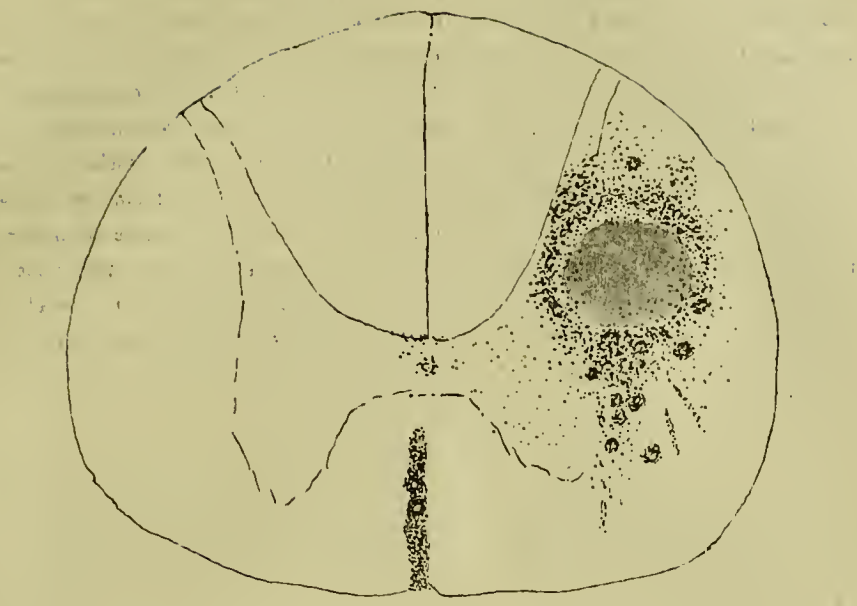

Fig. 10.—Transverse Section of the Spinal Cord, showing the position of the Gummatous Tumour and the surrounding Myelitis.

gumma and a solitary tubercle of the cord. There were no tubercles in any other part of the body, whilst there was a strong probability of exposure to the risks of syphilis, and the microscopical appearance was more in favour of a gumma than of tubercle; also in the pia mater the arteries in many sections showed well-marked endarteritis, and the adventitia was infiltrated with round cells (see Fig. 10). Some of the small veins presented endophlebitis, but the changes were not so marked as in the arteries. The walls of the smallest vessel were thickened both in the cord and in the meninges, and the vessels were surrounded by a zone of cell infiltration. The vascular changes in the meninges were best marked near the posterior nerve roots. Above the gumma there were well-marked tracts of ascending secondary degeneration in the posterior columns, and below it tracts of descending degeneration in the lateral columns.

Cases of intra-medullary tumour or gumma of the spinal cord, verified by post-mortem examination, are somewhat rare, and the symptomatology of the case recorded above presents several points of interest. The presence of pain for ten weeks before the onset of the numbness in the legs and paralysis was a point of diagnostic importance; so also was the steady progress of symptoms, and the fact that the anæsthesia at first was complete on one leg and only partial on the other.

Orlowsky (*Neurolog. Centbt.*, 1896, p. 1055) records a case of gumma of the spinal cord. The patient was a man aged 49 who had suffered from syphilis in 1887. He was treated energetically, but in 1892 a gumma of the tongue developed. In 1895 paresis of the legs developed. When examined there was œdema of the legs, contraction of the oblique abdominal muscles, atrophy and diminution of the electrical excitability of the muscles of the legs, increase of the reflexes and diminution of all forms of sensation in the legs, but more markedly so on the left side. There was a zone of hyperæsthesia at the level of the 5 to 10 ribs. Incontinence of urine and fæces. Death occurred suddenly. The autopsy revealed 2 gummata at the level of the 5 and 8 dorsal vertebræ—one in the dura, the second in the substance of the spinal cord. The cord was compressed and altered in form. There were syphilitic changes in the membranes and vessels.

Gowers (Diseases of the Nervous System, vol. 1, 2nd edition, p. 549) figures a section of the spinal cord showing a gumma occupying the position of the posterior horn and adjacent parts of the posterior and lateral columns in the lower cervical region. A second small gumma was present higher up. The symptoms were complicated by hemiplegia, &c., due to a gumma in the brain. Paralysis, with rigid flexor contraction of the left arm and leg, was apparently due to the growth.

Rosenthal—(*Klinik der Nervenkrankheiten, 2nd Aufl., Stuttgart*, 1875, p. 355) records a case of gumma of the meninges. The symptoms were neuraglic pain in the legs, followed by paraplegia, anæsthesia, analgesia, cystitis, and bedsore. The autopsy revealed a gumma of the spinal dura mater, of the thickness of the finger, and 3cm. long, which compressed the spinal cord on the left side from the 2nd to the 5th cervical vertebræ. A cerebral gumma was also present.

The *diagnosis* of a gumma of the cord or meninges is difficult, and is based on symptoms of a meningeal or intra-medullary tumour in a syphilitic subject. But as already mentioned, the diagnosis is rarely verified pathologically, either because the gumma, if present, is removed

by treatment, or because the symptoms have been due to some other syphilitic lesion. When symptoms of hemiparaplegia (Brown-Séquard's paralysis) develop in a syphilitic subject, a diagnosis of gumma is often made, especially if the unilateral symptoms remain stationary for some time. On rare occasions, such a diagnosis is proved to be correct; but usually either the symptoms disappear under treatment, or if a pathological examination is obtained, some other lesion is found.

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X. ANOMALOUS FORMS.

Syphilitic Hemi-paraplegia (Brown-Séquard's paralysis). —In spinal syphilis, not infrequently symptoms of hemi-paraplegia (Brown-Séquard's paralysis or unilateral lesion of the spinal cord) are present at an early stage of the disease. There is paralysis of one leg and anæsthesia of the other leg, with the additional symptoms produced by a unilateral lesion of the spinal cord. But in most cases the symptoms do not correspond exactly with those of a unilateral lesion. Frequently they would be better described as unequal affection of the two legs: one leg is markedly paralysed, the other only slightly; and as regards the sensory troubles they are most marked in the leg which is least paralysed. Also in most cases the stage in which symptoms of a unilateral lesion of the cord are present is of short duration, and soon both legs are markedly and almost equally affected. But occasionally a pure form of hemiparaplegia is met with, and bilateral paralysis and bilateral anæsthesia never develop. Considered from another standpoint, it is interesting to note that a considerable proportion of the cases of Brown-Séquard's paralysis hitherto recorded have been due to syphilis.

Oppenheim states that one-half to one-third of the cases of Brown-Séquard's paralysis which have come under his observation have been due to syphilis.

It is interesting to note that in many of the cases recorded, when the anæsthesia has not been complete, there has been unilateral loss of sensation to pain and

temperature, whilst sensation to tactile impressions has been normal or only slightly impaired.

Oppenheim points out that before or after the onset of the paralysis, he has observed irritative symptoms—tonic contraction of muscle on the side of the lesion, and pain on the side of the body opposite to the lesion. Thus in a case of lesion of the left half of the upper part of the cervical region of the cord, the symptoms were spastic paresis of the left arm and leg, with anæsthesia of the right side, but there were attacks of tonic contraction of the muscles of the left arm and leg, with severe pain in the limbs on the right side.

The following is a well recorded case, followed by post-mortem examination, published by Dejerine and Thomas (*Arch. de Physiologie Normale et Pathologique*, No. 3, 1898).

The patient was 36 years of age and had suffered from syphilis at the age of 16. The illness commenced with pain in the back, followed by pain in the left leg. At first the left leg only was paralysed; but later both legs were paralysed. The knee-jerk was increased on the left side; diminished on the right. There was no ankleclonus. On the left leg there was hyperæsthesia; on the right leg tactile sensation was only slightly impaired, but there was marked loss of sensation to pain and temperature. Incontinence of urine was present. At a later date a bed sore developed. On the left leg the hyperæsthesia was most marked for the sensation of cold. On the right leg there was only slight diminution of sensation for pain and heat, but marked diminution for cold.

At a still later date tactile sensation was normal on both legs, but there was loss of sensation to pain and temperature (both heat and cold) on the right leg; hyperæsthesia to cold on the left leg.

The post-mortem examination revealed a gummatous pachymeningitis, compressing and destroying a great part of the left half of the spinal cord between the fourth and sixth dorsal nerve parts. The posterior columns were almost unaffected. There was ascending degeneration in the left direct cerebellar tract, and in the column of Gowers, and descending degeneration in the left lateral pyramidal tract. At the seat of the compression there was a gummatous infiltration of the left half of the cord with periphlebitis and periarteritis (syphilitic meningo-myelitis and gummatous sclerosis).

Piatol and Cestan (abstract *Virchow's Jahresbericht*, 1897, p.570.) report

the case of a female, aged 28, who suffered from severe pain in the right leg two years after syphilitic infection; then flaccid paralysis followed. The right leg was hyperæsthetic. There was anæsthesia to pain and temperature on the left leg. At a later date incontinence of urine and a bed sore developed. Death occurred from pyæmic symptoms.

The autopsy revealed syphilitic meningo-myelitis in the upper part of the spinal cord, which had developed from the anterior and lateral vessels of the left half of the cord. On the right side the changes had involved the lateral tract and posterior horns; on the left side the anterior columns and anterior horn.

Jacobs (*New York Med.*, August 27, 1898) records the the case of a man, aged 40, who came under treatment at the John Hopkins Hospital Dispensary in February, 1893, on account of twitching of his toes at night, and loss of power in walking. He had contracted syphilis twelve months previously, and had suffered from secondary symptoms. Until about three weeks before he came to the hospital he considered himself quite well; then he first noted diminished sexual power. No other symptoms were noticed until about ten days later, when he observed loss of power in the right leg one morning.

"On attempting coitus, he completely failed. His lameness rapidly increased, and he had to resort to a cane, and with it he was only able walk with difficulty." There was no absolute loss of control of the sphincters, but for a few days he had to pass his water at once whenever there was a desire to micturate.

Examination showed that there was no paralysis of the left leg, but on the right side there was marked weakness in the flexors of the thigh, lower leg, and dorsal flexors of the foot; while the extensors of the thigh, lower leg, and plantar flexors of the foot, as well as the adductors, were nearly normal. The abductors and rotators were slightly weak. Sensation was normal on the upper half of the body, the right side of the abdomen, and right leg. On the *left* side, below the level of the umbilicus, sensory symptoms were present; tactile impressions were felt and localised, but, especially below Poupart's ligament and the gluteal fold, slight tactile impressions were not felt so sharply as on the right leg. Over the same area there was loss of sensation to pain and temperature. The knee jerk was increased, and there was both patellar and ankle clonus on the paralysed side (right). Under treatment with mercury and iodide of potassium there was marked improvement in a month, but sexual impotence persisted. At the end of two and a half months the motor and sensory symptoms had almost disappeared.

Nearly two years after the onset of the paralytic symptoms there was still loss of sexual power, and slight spastic gait on the right side. In December,

1896, it was noted: "He has not been able to have sexual intercourse, though desire for it is present. Since the first there has been no trouble whatever with his sphincters."

Mann has analysed the symptoms in six similar cases. In all of these the sensation to pain and temperature was absent, but that to touch was preserved, and the reverse conditions never occurred.

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Triplegia.—This is the name given to a variety of syphilis of the nervous system in which three limbs are paralysed. It is really a combination of paraplegia, from spinal syphilis, with hemiplegia from a cerebral lesion. In some cases the paraplegia occurs first, and the hemiplegia at a later date; in other cases the reverse order of development is met with.

The following is a good example of triplegia which has come under my observation at the Manchester Royal Infirmary. The patient was admitted under the care of Dr. Dreschfeld in 1896 with the following history. In June, 1895, he had begun to suffer from pains in the head. In October 1895, he was troubled with giddiness and pains in the back, and he was unsteady in walking. The legs gradually became weak until there was marked paraplegia and inability to walk. Incontinence of urine also developed. He had suffered from syphilis four years before the onset of the paraplegia.

On admission to the hospital in Feburary, 1896, there was paralysis of both legs. The knee jerks were increased

and ankle clonus was present on both sides. There was incontinence of urine and cystitis. Shortly after admission marked, paresis of the right arm and right side of the face developed. Thus three limbs were paralysed—both legs and the right arm. The right external rectus was also paralysed. The optic discs and the fields of vision were normal. At a later date the intercostals became paralysed and death occurred.

Cases simulating disseminated sclerosis.—A few cases are on record in which the symptoms of spinal or cerebro-spinal syphilis have somewhat resembled those of disseminated sclerosis: but the resemblence is not a close one, and it is the cases of atypical disseminated sclerosis which syphilis simulates more closely. Both syphilis and disseminated sclerosis give rise to symptoms indicating multiple lesions of the nervous systems, and in this point the symptomatology of the two affections agrees. But multiplicity of lesion is not sufficient evidence on which to base a diagnosis of disseminated sclerosis, and careful examination usually leaves little doubt as to the nature of the disease.

From the writings of Bechterew, Sachs, and others, it appears that the following points are of importance in the diagnosis between disseminated sclerosis and multiple syphilitic lesions of the nervous system. Nystagmus is very rare in cerebral syphilis, and the presence of this symptom is an indication strongly in favour of disseminated sclerosis. Intention tremor and scanning speech are also in favour of disseminated sclerosis. Sachs attaches the greatest importance to the ocular symptoms. Paralysis of ocular muscles may occur in both conditions, but they are rarely so complete in disseminated sclerosis as in cerebral syphilis. It is rare for ocular paralysis to preceed other symptoms for a year or so in disseminated sclerosis, but

this is much more frequently the case in cerebral syphilis. Immobility of the pupil—failure to react to both light and accommodation is distinctly in favour of syphilis (Sachs). This symptom is more common in syphilitic disease of the nervous system than in other affections. The presence of optic neuritis is in favour of syphilis; optic atrophy in favour of disseminated sclerosis (but there are exceptions to this general statement). The acute development of paraplegia or of other symptoms is in favour of syphilis: so also are oscillations in the intensity of symptoms, remission of symptoms for months or years, and improvement under anti-syphilitic treatment. The history of syphilis and the indications of past syphilitic disease are also important indications.

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1. Bechterew.—*Arch. f. Pyschratrie*, Bd. xxviii.
2. Sachs.—*Journal of Nervous and Mental Diseases*, 1897, p. 176.

Case Presenting the Symptoms of Primary Lateral Sclerosis. Spinal syphilis is sometimes met with, in which the symptoms are those of spastic paraplegia, without any affection of bladder or rectum, and without affection of sensation. There is then a strong resemblance to the cases, which have been described as primary lateral sclerosis, but in which post-mortem examination almost invariably proves that other parts besides the lateral pyramidal tracts are affected. A careful history of these syphilitic cases usually shows that symptoms indicating affection of other parts of the cord, have been present at some period—such as pain in the back or limbs, bladder symptoms, &c. Or if the case has been carefully followed such symptoms have developed in the further course of the disease.

Minkowski records a case in which symptoms of lateral

sclerosis followed syphilitic infection. There was no affection of sensation. The case is recorded as one of primary lateral sclerosis, but the autopsy revealed sclerosis of the crossed pyramidal and direct cerebellar tracts.

Minkowski.—*Deutsch. Arch. f. klin. Med.*, Bd. 36, p. 443.

Cases presenting the Symptoms of Anterior Polio-myelitis.—Goldflam records a case which clinically presented the symptoms of sub-acute anterior polio-myelitis, viz.: flaccid paralysis of the legs, with atrophy of the muscles, diminution of the electrical excitability, disappearance of the tendon reflexes, persistence of the skin reflexes, absence of anæsthesia, absence of bladder or rectal disturbances. These symptoms occurred one year after the syphilitic infection, and during the presence of undoubted syphilitic ulceration of the throat. No cause could be discovered which would account for the symptoms except syphilis, and under anti-syphilitic treatment recovery occurred. Only a few similar cases are on record. Rumpf reports a case in which the symptoms corresponded in most points with sub-acute anterior polio-myelitis, but the knee jerks were present.

The anterior horns of grey matter receive their blood supply from the anterior median arteries, which are branches of the anterior spinal, and as syphilis is so liable to produce disease of the blood vessel, it is conceivable that occasionally the branches of the anterior spinal system of arteries may be chiefly affected, and that anterior polio-myelitis may result.

As regards the diagnosis, the syphilitic history, and the improvement under anti-syphilitic treatment are the chief indications.

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Cases simulating forms of muscular atrophy—(Aran-Duchenne type, amyotrophic muscular atrophy, idiopathic muscular atrophy, &c.)

Cases of syphilitic cervical pachymeningitis, causing atrophy of the muscles of the hand, have been already referred to (sec. V.) Progressive muscular atrophy and amyotrophic lateral sclerosis may occur in patients who have previously suffered from syphilis, but since more frequently there is no specific history, and no signs of previous specific disease, these forms of muscular atrophy cannot be attributed to syphilis in the few cases in which there happens to have been a previous specific infection.

On the other hand, there are cases of distinct spinal syphilis on record which, have somewhat resembled progressive muscular atrophy, or amyotrophic lateral sclerosis in their symptomatology; but the resemblance has not been close, and a careful examination has usually revealed symptoms which have shown clearly that the case was not one of the diseases just mentioned. Thus, for example, in the syphilitic cases, careful examination reveals sensory troubles, objective and subjective, or affection of the bladder and rectum, or diplopia and ocular affections, or vertigo and cerebral troubles, *i.e.*, symptoms which do not occur in amyotrophic lateral sclerosis, in progressive muscular atrophy, or in idiopathic muscular atrophy. In the cases of spinal syphilis characterised by muscular atrophy, one or other of the group of symptoms just mentioned is rarely absent; also the beneficial effect of anti-syphilitic treatment is most

important evidence, because in the non-syphilitic cases the progress of the disease is not arrested by treatment.

Raymond.—*Soc. med. des hôp. Février 9th, 1893.*

Gilles de la Tourette.—*Myélites Syphilitiques,* Paris, 1899, p. 46.

There are many other rare forms of spinal syphilis which it is almost impossible to classify. Thus in syphilitic meningitis, involving the lower part of the cord, the gait and the manner in which the patient rises into the erect posture, may be almost exactly the same as in *pseudo-hypertrophic paralysis and idiopathic muscular atrophy,* but the severe pain and sensory symptoms are diagnostic. (see section V.)

In cases of spinal syphilis, I have not infrequently found the sensory symptoms to consist chiefly of loss of sensation to pain and temperature, whilst tactile sensation has been normal. Formerly these sensory symptoms were thought to indicate syringo-myelia, but it has been shown clearly that in many other diseases a similar sensory affection is met with. In the syphilitic cases the diagnosis is usually easy; the onset of the symptoms is usually more rapid, and there are generally indications of previous syphilitic infection.

Cavities in the Spinal Cord.—Wullenweber reports a case, in which post-mortem examination revealed a cavity in the cord, evidently secondary to a syphilitic meningitis.

The patient was a woman, aged 28, who had previously suffered from syphilis. The illness commenced with pain in the back (lumbar region). This was followed by headache, and then by weakness and rigidity of the legs. The patellar reflexes disappeared. At a later date painful spasmodic contractions often occurred in the right leg, and later still, girdle pains became very troublesome.

The legs finally became atrophied and completely paralysed, and bedsores and paralysis of the sphincter developed.

Pathological examination of the spinal cord revealed a central cavity, extending from the lower lumbar to the upper cervical regions. The cavity was localised to the grey matter. There was marked syphilitic meningitis and syphilitic diseases of the blood vessels. The case was not one of true syringomyelia; the cavity was apparently due to a necrosis of the spinal cord, caused by syphilitic disease of the vessels. The symptoms and clinical course also were not those of syringomyelia.

Experiments on animals have shown that necrosis of the grey substance of the spinal cord can be produced by temporary ligature of the aorta, whilst the white substance is able to resist for a longer period.

In Wullenweber's case circulatory disturbances, and the anæmia produced thereby, were the result of marked endarteritis and of the compression of blood vessels by the cicatricial tissue in the meninges. The circulatory disturbances had produced nutritional changes, and these had been followed by the formation of a cavity.

Schwarz also carefully records a similar case of syphilitic meningo-myelitis, with the formation of a number of cavities in the grey matter of the spinal cord. He discusses carefully the relation of the changes to the arterial supply. The cavities appeared to arise in the middle of homogenous material imbedded in the grey matter.

References.

1. Schwarz.—*Zeitschrift f. klin. Med. Bd.* 34., p. 469.
2. Wullenweber.—*Münchener med. Woch.*, Aug. 9, 1898.

Lesion of the cauda equina.—A specific meningitis, gummata, or gummatous infiltration may affect the cauda equina and cause adhesion of the nerve roots with one another and with the meninges. The symptoms which have been observed have been :—pain in the region of each sciatic nerve (double sciatica), and in the region of the bladder, rectum, and genital organs ; anæsthesia in the gluteal region, down the back of the thighs, and in the area of distribution of the sacral plexus, the exact distribution varying with the extent of the lesion. Paralysis of the bladder and rectum has also been observed. Oppenheim mentions two cases in which improvement occurred under anti-syphilitic treatment.

References.

1. Oppenheim.—*Lehrbuch der Nervenkrankheiten. Zweite aufl.*, Berlin, 1898, p. 239-40.
2. Osler.—*Journal of Nervous and Mental Diseases*, 1889.

Neuritis of Nerve Roots.—Kahler has recorded a case of cerebro-spinal syphilis, in which neuritis of various spinal and cerebral nerve roots was found on pathological examination. (Abstract *Virchow's Jahresbericht*, 1887, Bd. II., p. 156 and 654).

A few other cases are on record.

XI. SYPHILIS AND TABES AND PSEUDO-TABES.

Syphilitic Pseudo-Tabes.—Spinal syphilis occasionally gives rise to symptoms which, at least in one stage of the disease, resemble, in some points, those of locomotor ataxia; also cases are occasionally met with, in which, at first, the symptoms have been attributed, even by able neurologists, to locomotor ataxia, and yet in course of time they have disappeared for the most part, and practically recovery has occurred. Probably, in such cases, the symptoms are due to a syphilitic lesion of the meninges, invading the posterior columns of the cord, and the posterior nerve roots.

Such a syphilitic lesion may cause loss of knee jerks, ataxia, shooting pains, and sensory disturbances in the legs, &c. The spinal disease may be associated with cerebral syphilitic lesions, *e.g.*, basal specific meningitis, and specific affection of the cranial nerves. The cerebral lesions may produce paralysis of ocular muscles, immobility of the pupils, affection of the auditory, trigeminal nerves, or other cranial nerves. As all these symptoms may occur in true tabes, the diagnosis is occasionally difficult. But usually a careful consideration of the case enables its true nature to be recognised.

Diagnosis.—In the syphilitic pseudo-tabes, the symptoms, as a rule, develop more rapidly than in true tabes. In the pseudo-tabes there are frequent oscillations in the severity of the symptoms; the knee jerks often show remarkable variations—at one time being absent, at

another time present. Often there is paresis or paralysis of the limbs, whilst this symptom only occurs at the last stage of true tabes. There is often a tendency for one side to be more affected than the other in pseudo-tabes; one leg may be more ataxic than the other; one knee jerk or one eye may be more affected than the other (B. Sachs).

In syphilitic pseudo-tabes, optic neuritis may occur; in true tabes when the optic diseases are affected, the change is one of *primary* optic atrophy. The pupils are often immobile in pseudo-tabes, and gastric and other crises very rarely occur. The symptoms do not show that progressive tendency which is so common in true tabes. Also in the pseudo-tabes there is often marked improvement under anti-syphilitic treatment, and sometimes there is almost complete recovery.

A few cases are on record in which the difficulty in diagnosis has been very great, and a correct opinion could only be formed after carefully watching the symptoms for a long period.

The following are brief notes on a case which I have seen on several occasions during the last fifteen years at the Manchester Royal Infirmary. He was first a patient under the care of Dr. Ross, in 1884, at the time when I was House Physician. From the absence of his knee jerks, and the presence of numbness in the legs, he was thought to be suffering from early locomotor ataxia. I saw the patient last when he was under the care of Dr. Harris, in 1898.

W. W., in 1864, suffered from syphilis. He was then 20 years of age.

In 1883 slight symptoms of bulbar paralysis developed.

In 1884 he suffered from gummatous ulcerations on the scalp and legs. On admission to the Infirmary, in that year, he complained of *numbness in the legs*. The *floor felt soft* to the feet. There was slight impairment of sensation to pain in the legs. He was able to walk and there was no ataxia;

no Rhomberg's symptom. The muscular sense was normal and there was no paralysis, but both *knee jerks were absent*. The voice had a nasal twang and certain letters were not pronounced clearly. There was a little difficulty in swallowing, and a sensation of numbness at the back of the throat. But there was no actual facial paralysis, and the tongue movements were good. A syphilitic ulcer was present in the anterior tibial region of one leg. The symptoms just mentioned, though not diagnostic, were suggestive of early locomotor ataxia.

In 1891 the slight bulbar symptoms had disappeared, but the other nervous symptoms remained.

At a later date (1892) symptoms and signs of aortic stenosis were detected, and in 1898 he was under the care of Dr. Harris for the cardiac affection.

In 1898, when I last examined him, he was able to walk quite well. There was no ataxia and no Rhomberg's symptom, but both knee jerks were absent (even when tried according to Jendrassik's method). There was no loss of power in the limbs, and no affection of sensation—the numbness present in 1884 having entirely disappeared. There were no bulbar symptoms and no paralysis of ocular muscles. The pupils were unequal, the right being larger than the left. Both pupils reacted to accommodation. The right reacted to light quite well; the left feebly. There was no pain in the back or legs, and no girdle sensation.

The absence of knee-jerks (in 1884), the numbness in the legs, the sensation in the feet as if the floor were soft, the history of syphilis, and the presence of specific ulcers were very suggestive of beginning locomotor ataxia. But sufficient time has now elapsed to show that the case was one of syphilitic pseudo-tabes. The nervous symptoms had all disappeared at the end of 14 years, except the absent knee-jerks.

The following are details of several cases in which symptoms of tabes have been associated with syphilitic lesions in the spinal cord.

The cases tabulated show, that symptoms very similar to those of true tabes may be produced by syphilitic meningitis or gummatous meningitis, with extension of the inflammatory changes to the posterior columns (as in the first three cases).

In other cases pathological examination reveals the early changes in the posterior columns which are characteristic of tabes, along with a syphilitic meningitis and syphilitic vascular changes.

Author.	Journal.	Symptoms.	Pathological Changes.
Eisenlohr.	*Virchow's Jahresberichte* 1889, Bd. ii., p. 95, (Abstract.)	Shooting pains for many years; knee jerks sometimes present, sometimes absent. Marked paresis of the legs several weeks before death.	Turbidity of pia-mater on the posterior surface of the spinal cord; several small gummata in the pia, the largest at the level of the 8th dorsal vertebra. At this level marked degeneration of the posterior columns, and a little of the adjacent lateral columns. The posterior roots were degenerated at this region also. Degeneration in the cord extended for 1½ cm. Below the cord was normal. Scattered degenerated fibres were found in the posterior columns above the part mentioned.
Oppenheim.	*Berliner Klin. Woch.*, 1888, p. 1061.	Absent knee jerks; shooting pains; absence of pupillary reflex; sensory disturbances: paralysis of R third nerve; paresis of the L; bulbar symptoms; paralysis of vocal cords; attacks of vomiting and coughing; difficulty in swallowing; paralysis of the spinal accessory nerve, and of the soft palate. Later, knee jerks returned and ankleclonus developed.	Chronic internal pachymeningitis and gummatous arachnitis in the lumbar and lower dorsal region; membranes adherent to the cord at this region; invasion of posterior columns.
Ewald.	*Berliner Klin. Woch.*, No 12, 1893.	Absent knee jerks: Rhomberg's symptom; ataxia; inequality of pupils and loss of light reflex; loss of sensation to temperature and pressure; inability to distinguish between the head and the point of a pin on the legs.	Changes not characteristic of tabes; chronic fibrous and gummatous spinal arachnitis; invasion of the posterior columns of the cord by the inflammatory changes; chronic interstitial myelitis; endarteritis and phlebitis.
Sachs.	*New York Med. Jour.*, Jan. 6th, 1894.	Typical symptoms of tabes.	Posterior sclerosis associated with syphilitic lepto-meningitis, invading the cord at different levels; syphilitic arteritis.
Dinkler.	*Deutsche Zeitschrift f. Nervenheilkunde* (Abstract *Neurol. Centralblatt*), 1893, p. 406.	Shooting pains in legs; girdle sensations; sensory symptom in legs, but knee jerks *present*, R. > L.	Changes in posterior columns indicating early tabes, along with syphilitic spinal arachnitis; endarteritis.
Kuh.	*Arch. f. Psych.* Bd. 22, 1891, p. 699.	Knee jerks absent; ataxia; impotence; paræsthesia; Rhomberg's symptom. Tabetic affection of joints; sluggish pupillary reflex.	Degeneration of posterior columns characteristic of locomotor ataxia; also meningitis from cerebrum to cauda equina; syphilitic disease of the vessels; granulation tumours in walls of vessels; end-, peri- and mesarteritis. Tabes with syphilitic meningitis.

Author.	Journal.	Symptoms.	Pathological Changes.
Valentin.	*Neurologisches Centralblatt*, No. 1, 1899. p. 45 (Society Reports).	Gastric crises; diplopia; difficulty in walking; vertigo. Retention and incontinence of urine; 2 years later sudden paralysis of all four limbs; ataxia. Left knee jerk lost, right increased. Pupils L. > R.; no reaction to light.	Cervical meningitis. In the upper part of the spinal cord the whole transverse area diseased. Degeneration of the periphery of the cord; sclerosis of the lateral columns in the dorsal region; beginning degeneration in the posterior columns of the lumbar region. Syphilitic disease of the arteries. The author regards the case as one of early tabes combined with syphilitic menin-go-myelitis.

Syphilis and Locomotor Ataxia.—The relation of syphilis to locomotor ataxia is a question which has been much discussed, and even at the present time different opinions are held. Many authors (Fournier, Erb, Gowers, Vulpian, Grasset, and others) maintain that syphilis is the cause of a large percentage of cases of locomotor ataxia. Some observers remain undecided on this question (Virchow and others): whilst a few (Leyden and others) hold that syphilis does not cause locomotor ataxia.

Gowers states that of 50 consecutive cases of the disease in men, seen in private consulting practice 29 (*i.e.* 58 per cent.) gave a history of chancre known to be hard, or of secondary symptoms: and eight others had a venereal sore of unknown nature. He points out that the ascertainable facts are certainly below the real facts. When a deduction is made for possible accidental coincidence, he thinks that there is a causal relationship between syphilis and tabes in from one-half to three-quarters of the cases.

Storbeck has recently published statistics of Prof. Leyden cases in which a history of syphilis was much less frequent, but the following are the last statistics

published by Professor Erb on this subject. (*Berliner klin. Woch*, No. 11, 1896).

PROF. ERB'S 200 CASES OF TABES.

	First Hundred.	Second Hundred.	Mean p. c.
1. Cases without evidence of syphilitic infection	5	10	7·5
2. Cases with indications of previous syphilitic infection	95	90	92·5
(*a*) Cases with a definite history of secondary syphilis	60	63	61·5
(*b*) Cases with history of a chancre, but without any history of secondary symptoms	35	27	31·0

In 25 of the 62 cases (*b*) in which there was a history of a chancre, but no secondary symptoms, either the chancre was of the hard variety, or it was regarded as syphilitic, and mercury or potassium iodide were prescribed by the medical attendant. Of the 15 cases in which there was no evidence of syphilis, in 11, from the history of gonorrhœa, stricture, bubo, repeated abortion, &c., there was a possibility of the patient having been exposed to the risks of syphilitic infection. In only 4 cases out of the 200 tabulated (*i.e.*, 2 per cent.) could Prof. Erb exclude with certainty the possibility of exposure to the risks of syphilitic infection.

As regards its pathological anatomy, locomotor ataxia is not a true syphilitic affection, *i.e.*, the changes in the cord do not resemble changes of a syphilitic nature which occur in other organs. Also the cord changes in tabes are quite different from the lesions in spinal syphilis. By those who support the syphilitic origin of locomotor ataxia the changes are regarded as a post-syphilitic

degeneration, and are supposed to be due to a toxin produced in the system by the syphilitic infection.

It is a curious fact, that cases of tabes are seldom met with, in which indications of tertiary syphilitic lesion of other organs are present.

On post-mortem examination, evidence of syphilitic disease in the viscera is rare; still cases are on record; and there are also a few in which the brain or cord has presented syphilitic changes in addition to the degeneration characteristic of tabes in the posterior columns (see Nonne *Berliner klin. Woch.*, No. 15, 1899). As exact statistics are wanting, of the proportion of syphilitic changes found in the post-mortem room in a large series of cases of persons dying of various diseases, (*i.e.*, the proportion of cases of accidental syphilitic lesions), it is difficult to draw conclusions as to the value of the fact just mentioned. Westenhoeffer found undoubted syphilitic changes recorded in 15 out of 61 autopsies on cases of tabes at Virchow's Pathological Institute.

It is said that prostitutes seldom suffer from tabes, and that in countries where syphilis is exceedingly common, as for example Bosnia, Herzegovina, and Kirghiz in Central Asia, locomotor ataxia is very rare. But it is probable that exact statistics on these points would be very difficult to obtain. It is also interesting to note that Reumont found only 40 cases of tabes in 3,600 persons who had suffered from syphilis—*i.e.*, only 1·1 per cent.

Undoubtedly in a small proportion of tabetic patients no evidence of past syphilis can be obtained.

On the other hand, there can be no doubt about the much greater frequency of a history of syphilis and venereal disease in tabes than in many of the other chronic disease of the nervous system, as for example, disseminated sclerosis or amyotrophic lateral sclerosis;

and it is very probable that there is some connection between tabes and venereal disease.

It is a striking fact that very frequently the syphilitic symptoms have been very mild, and it is very rare to obtain a history of *severe* syphilis in a case of tabes.

In a considerable number of tabetic patients in Manchester I have obtained a history of a chancre, but no other symptoms had been observed. In some of these cases, as far as one can judge from the history, the chancres have been of the soft variety, and Hitzig has suggested the possibility of a virus, capable of commencing the tabetic changes, being associated with a soft chancre.

Though tabes may be regarded as a post-syphilitic degeneration in the majority of cases, it can scarcely be looked upon as a form of spinal syphilis in the strict sense, and hence it is not necessary to consider the disease further.

XII. PROGNOSIS.

The prognosis in spinal syphilis is, on the whole, better than in other chronic diseases of the cord, and also better than in the cerebro-spinal syphilis.

The prognosis differs according to the form of the affection, and it has been considered already under the descriptions of the special varieties.

Another important point is the extent of the involvement of the cord. If only the meninges are affected, the prognosis is better, but if much of the transverse area of the cord is involved at any level, the prognosis is worse. In such cases, even if the affection should subside, sclerotic changes may remain, which cannot be removed by treatment.

The prognosis is worst in the cases of acute paraplegia ("acute myelitis"), especially when there is complete paralysis of the legs, with affection of the bladder and rectum, (see p. 57). Some of these cases rapidly terminate fatally. In many cases of meningo-myelitis, the condition varies from time to time; improvement may occur, or the symptoms may long remain stationary, or they may advance. The more marked the paraplegia, and the longer it persists, the worse the prognosis. In a lesion of the lumbar region, the prognosis is worse than when the dorsal region is affected.

As already mentioned, complete paralysis of the bladder, cystitis, or bedsores are very bad indications, and death often occurs from these complications. Paralysis of the intercostals, or the development of pneumonia are also very serious symptoms.

In the 32 cases in Manchester classified on page 11, death occurred in 9, recovery in 10. The nature of the fatal cases was as follows :—Five cases of "acute myelitis," one of Erb's syphilitic paralysis, one of gumma in the cord, one of triplegia, and one of meningo-myelitis in which changes were found in the posterior and lateral columns (see page 86).

The nature of the ten cases which recovered was as follows :—Five of meningo-myelitis, three of meningitis, one of "acute myelitis," and one of pseudo-tabes.

In two of the cases of meningo-myelitis which recovered, there was only paresis of the legs with slight bladder symptoms ; in one case there was complete paralysis of the bladder, with paresis of the legs : in one case paraplegia was well marked. In one of my cases there was retention of urine for a few weeks, with marked paraplegia for five months ; but under anti-syphilitic treatment complete recovery occurred, and in course of time the patient could walk quite well. A year afterwards right-sided facial paralysis and paralysis of the right third nerve developed. Recovery again occurred under treatment, but, later, left-sided ophthalmoplegia developed. In another case symptoms of acute myelitis developed, there was marked paraplegia for about ten weeks, with retention of urine and cystitis ; the symptoms subsided, and the patient gradually recovered. He was able to follow his employment eighteen months afterwards.

In the other cases (13 out of 32) either the disease remained stationary, or varied from time to time, and the patient passed from under observation.

XIII. TREATMENT.

At the early stage of spinal syphilis, especially in cases where the meninges only are involved, treatment is of the greatest importance. It is then that we may hope to arrest the syphilitic changes, and to produce the greatest effects. When the cord has been seriously damaged, and nerve elements destroyed and replaced by cicatricial tissue, we cannot hope, as Sir Wm. Gowers points out, to remove this scar tissue by treatment. Hence early diagnosis is of the greatest value.

In individuals who have suffered from syphilis, it is important to recognise the early or premonitory symptoms of spinal syphilis, and at once to commence vigorous treatment.

In all cases anti-syphilitic treatment should be thoroughly carried out, and it is best to combine mercury and potassium iodide. The form which is most commonly prescribed is the combination of mercurial inunctions with potassium iodide internally. A drachm or more of the blue (mercurial) ointment may be rubbed into the skin daily, until the patient has been brought well under the influence of the drug, and the ointment continued in quantities sufficient to maintain the action, but to avoid the toxic effects. The ointment may be rubbed into the skin of the limbs in succession (one limb each day). It is well to rub the ointment downwards, towards the distal part (*i.e.*, in the direction of the hairs). When the inunctions are made in this direction, irritation of the hair follicles is less likely to occur. It is, of course, necessary to watch for the development of toxic symptoms.

Mercury may be given in the form of pill, powder, or mixture, if preferred, and when it is necessary to bring the patient rapidly under its influence, or when the patient is not likely to carefully follow out the inunction treatment, then subcutaneous or intravenous injections be employed.

Hutchinson recommends the grey powder (Hydrargyrum cum cretâ) in small doses for a long period. He advises one grain of this powder in a pill combined with opium ($\frac{1}{5}$ to $\frac{1}{4}$ of a grain), so as to avoid diarrhœa at the onset of the treatment. He prescribes this pill four, five, six, or seven times a day, and thinks that by dividing the doses, toxic symptoms are avoided, and the desired effect produced with much greater certainty.

The two important evil effects of mercury are salivation and diarrhœa; the latter can be avoided by the use of opium, the former by allowing the patient to suck a small piece of alum for a short time every day, or by the use of an alum mouth wash, and by keeping the teeth well cleaned. With care, it is possible to continue a thorough mercurial treatment, for a long period without causing any bad effects.

It is well to interrupt the mercurial treatment, from time to time, for a few weeks. If recovery should occur, or if the symptoms remain stationary, it is still well to advise a mercurial course for a short time once or twice a year.

It is advisable to give *iodides* in all cases. It is best to commence with a small dose at first and then to increase the amount. Five or ten grains of the iodide of potassium or sodium may be given three times a day at first, and then the dose may be increased to 10 or 20 grains, or even a much larger quantity.

Occasionally iodides are badly borne, even 10 grain doses cannot be taken without causing great gastric irritation

or other symptoms. I have found in this case that 5 grains (followed by a little milk) every two hours has been easily taken, and thus 30 grains could be given in the 24 hours without any bad effects.

When mercurial inunctions and iodides have been given for a considerable time, the two drugs may be used alternately—the iodides being discontinued during the treatment by mercurial inunctions and vice-versa.

Iodides are often given with aromatic spirits of ammonia, and Hutchinson attaches great importance to the combination: he believes the good effect of the iodides to be increased by the addition of ammonia.

Often a combination of the three iodides, of potassium, sodium, and ammonium, along with aromatic spirits of ammonia, answers very well. Hutchinson thinks that, as a rule, it is not well to give iodides continuously for a long time. He recommends that the drug should be omitted for a few days at a time and then commenced in a smaller dose. Sir William Gowers also believes that in 6 to 10 weeks anti-syphilitic treatment, especially iodide of potassium, "will effect all that it can achieve in the removal of the syphilitic process." He regards the long continued treatment by mercury and iodides as a great mistake, and thinks that by the continued use of potassium iodide, for months or years, the tissues of the patient may become so accustomed to its presence that the drug no longer holds check on the syphilitic processes.

After the symptoms have subsided it is advisable to give a course of anti-syphilitic treatment for a short time every year. Sir William Gowers recommends that for 5 years the patient should have, each year, a three weeks course of treatment of 20 or 30 grains of iodide a day.

It is important that the patient should have good nourishing food. If the symptoms should partially or

completely subside, exposure to cold, over-strain, and sexual excess should be avoided. Marriage ought to be forbidden, even when the course of the spinal syphilis has been favourable. The use of electricity—galvanic current—is probably of some slight service when legs are paralysed and flaccid. Gentle massage and passive movements of the legs are also helpful.

A warm bath daily is advisable, whilst the patient is under mercurial treatment.

Complications must be treated on general principles and need not be considered here; but a few words may not be out of place with reference to two of the most important, *i.e.*, cystitis and bedsores.

In a large number of cases, as already mentioned, paralysis of the bladder is followed by cystitis, pyelonephritis, septic poisoning, and death results therefrom. If cystitis can be prevented, in time the cord may recover. I have seen a number of cases in which the cord symptoms have gradually subsided, but death has occurred from the complicating cystitis and pyelonephritis. Hence in the treatment of spinal syphilis, one of the most important points is to try and prevent or diminish cystitis. When there is retention of urine, the catheter should be passed at least twice a day, and the bladder emptied. It is important to examine the patient's abdomen daily, just above the pubes, in order to feel if the bladder is distended, if there should be any indication or suspicion of commencing paralysis of the bladder. This is a point of some practical importance, because the patients often state that they pass their urine freely, but the micturition may be only dribbling from an over-distended and paralysed bladder.

When the catheter is passed, it is of the greatest importance that it should be perfectly aseptic. There

can be no doubt that it is extremely difficult to prevent cystitis when the bladder is paralysed, however careful the attention to strict cleanliness of the catheter may be; but on the other hand, it is quite certain, that by want of cleanliness, cystitis is often excited or increased.

A soft india-rubber catheter is best for general use. Before being passed, it should be disinfected well by carbolic lotion or some other antiseptic. Mr. Wright recommends that it should be placed in hot diluted Jeyes' fluid, and that the orifice of the urethra should be sponged over with Jeyes' fluid before the catheter is used. These precautions are important whenever a catheter is passed, but are particularly so when the bladder is paralysed. After being used, the catheter should be carefully washed and placed in antiseptic solution till used again. In order to prevent the lodging of any septic matter, just at the tip of the catheter, beyond the eye, it is best to use a catheter which is solid at this part, (*i.e.*, solid at the tip just beyond the eye).

One catheter should not be used too long; when it becomes damaged in any way it should be at once replaced by a new one.

When cystitis has actually developed, the bladder should be washed out twice a day with boracic acid lotion (15gr. to the ounce of water) made luke-warm just before use by the addition of a little hot water; or a solution of sodium salicylate (30gr. to the ounce of water) may be used in the same way. Boracic acid and salol are often given internally in such cases.

When there is constant dribbling of urine, a porcelain bed urinal may be employed for male patients; but it sometimes causes irritation and œdema of the end of the penis in constant contact with it, and occasionally sloughing occurs. In such cases the genital organs may be

surrounded by some absorbant antiseptic wool, which is changed frequently.

Another point of great importance, as already pointed out, is the prevention of *bed sores*, which frequently cause death through septic absorbtion. In all cases of spinal syphilis, when the patient is obliged to remain in bed for a long period, the greatest cleanliness is necessary. The position should be frequently changed, and cotton wool should be used to relieve pressure. But it is always best when there are signs of irritation of the skin, to place the patient on a water bed. If the skin over the sacrum should become red, it may be washed with spirit lotion, or a lotion containing 10 grains of tannin in ℥i of rectified spirit, and the bed sheet may be dusted with oxide of zinc or starch powder, at the part where the gluteal region will come in contact with it. When the skin is once excoriated, zinc ointment, carbolised vasiline, boracic, or iodoform ointment may be used. When the bed sore is deep and sloughing, iodine and starch paste may be employed (for formula see Martindale and Westcott's Extra-Pharmacopæia, 1898 p. 282).

In many cases of spinal syphilis, considerable improvement follows careful treatment; sometimes brilliant results are obtained, and complete recovery occurs. On the other hand there are many cases in which treatment has little influence, and the cases of syphilitic paraplegia of a very acute onset (so called acute myelitis) often run a rapid course and terminate fatally in spite of the most vigorous treatment.

Nothwithstanding the favourable course and good results of treatment in many cases, there still remains a large number of cases, in which, at the best part of life, a previous healthy man is incapacitated for a long period or permanently; and in other cases, fortunately less

common, a fatal termination rapidly occurs. And yet, syphilis is a preventable disease. This is not the place to discuss the question of the legal control of the contagious diseases, nor the prophylaxis of syphilis, which is so easily expressed in the one word—morality. Apart from all higher motives, and looked at simply from the physical standpoint, can there be any stronger argument for morality than the miserable picture of a man, at the prime of life, suffering from incurable paraplegia, with paralysis of bladder and rectum and a large sacral bed sore?

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